copy 4

JAN 6 1937

Volume X December, 1930 Number 6

PROPERTY OF
DENTAL LIBRARY
UNIVERSITY OF MICHIGAN
DON'T MUTILATE OR REMOVE

THE JOURNAL OF DENTAL RESEARCH

(Continuing the Journal of the Allied Dental Societies)

EDITED BY

J. L. T. APPLETON, JR.
Philadelphia
ELMER S. BEST
Minneapolis
THEODORE B. BEUST
Louisville
ARTHUR D. BLACK
Evanston
JAMES R. BLAYNEY
Chicago
C. F. BÖDECKER
New York City
R. W. BUNTING
Ann Arbor
H. E. S. CHAYES
New York City
FRANK COLYER
London
KAETHE W. DEWEY
Pittsburgh
MARTIN DEWEY
New York City
F. N. DOUBLEDAY
London
WM. B. DUNNING
New York City
HENRY W. GILLETT
New York City
BERNHARD GOTTLIEB
Vienna
WILLIAM K. GREGORY
New York City
EDWARD H. HATTON
Chicago
MILO HELLMAN
New York City
PERCY R. HOWE
Boston
THADDEUS P. HYATT
New York City
PHILIP JAY
Ann Arbor
E. V. MCCOLLUM
Baltimore
FREDERICK S. MCKAY
New York City
JOHN A. MARSHALL
San Francisco
ARTHUR H. MERRITT
New York City
L. M. S. MINER
Boston
FREDERICK B. NOYES
Chicago
BALINT ORBAN
Vienna
WESTON A. PRICE
Cleveland
THEODOR ROSEBURY
New York City
F. E. RODRIGUEZ
Washington
KURT H. THOMA
Boston
M. L. WARD
Ann Arbor
B. W. WEINBERGER
New York City
WM. H. WELKER
Chicago
J. LEON WILLIAMS
New York City

AND BY REPRESENTATIVES OF AFFILIATED SOCIETIES

First District Dental Society of the State of New York (which in 1906 established the Journal of the Allied Dental Societies, now the oldest non-commercial journal in dentistry): JOHN OPPIE MCCALL

New York Academy of Dentistry: MALCOLM W. CARR

Robert R. Andrews Society for the Promotion of Research (Tufts): CHARLES E. HATCH

Harriet Newell Lowell Society for Dental Research (Harvard): HAROLD E. TINGLEY

William Jarvie Society for Dental Research (Columbia): JOSEPH SCHROFF

Dental Educational Council of America: H. E. FRIESELL, Pittsburgh

American College of Dentists: ALBERT L. MIDGLEY, Providence

International Association for Dental Research—

Sections (Secretaries):

Ann Arbor—U. G. RICKERT
Boston—FRED R. BLUMENTHAL
Chicago—RUDOLF KRONFELD
Cleveland—SAMUEL W. CHASE
Halifax—GEORGE K. THOMSON
Minnesota—CARL W. WALDRON
New Haven—WM. G. DOWNS, JR.
New York—BISSELL B. PALMER
Philadelphia—CARRIE KIRK BRYANT
Pittsburgh—LAWRENCE E. VAN KIRK
St. Louis—CLARENCE O. SIMPSON
San Francisco—JOHN S. SHELL
Toronto—A. J. BROUGHTON
Vienna—EMERICH KOTANYI
Winnipeg—E. ROY BIER

Association (General Secretary): WILLIAM J. GIES, *Executive Officer of the Board of Editors*

PUBLISHED BI-MONTHLY
AT MT. ROYAL AND GUILFORD AVENUES, BALTIMORE, MD.
BY THE JOURNAL OF DENTAL RESEARCH, INC.
EDITORIAL OFFICE 632 West 168th St., New York City
Copyright, 1930, by The Journal of Dental Research, Inc.

Entered as second-class matter June 24, 1919, at the Post Office at Baltimore, Maryland, under the act of March 8, 1879

Made in United States of America

La pratique stomatologique. VII. Orthodontie. By G. IZARD, Professeur d'Orthodontie à l'Ecole Francaise de Stomatologie; Stomatologiste des Hôpitaux de Paris. 1930: pp. 762; 6¼ x 3¼ in.; 714 illustrations; broché, 95 fr., relié, 110 fr. Masson et Cie, Paris, France.

Dentures et dentiers complets. By F. ACKERMANN, Ancien chef démonstrateur du Professeur Gysi, Université de Zurich; ancien assistant à l'Institut Dentaire et à la Faculté de Medicine de Genéve. 1930: pp. 538; 7½ x 4¾ in.; 538 illustrations; broché, 135 fr., relié, 150 fr. Masson et Cie, Paris, France.

Orthodontic laboratory manual. By GEORGE M. ANDERSON, D.D.S., Professor of Orthodontia, Baltimore College of Dental Surgery, University of Maryland, Baltimore. 1930: pp. 56; 7 x 4¼ in.; 23 illustrations; $2.00. C. V. Mosby Co., St. Louis, Mo.

Proceedings of the Seventh Annual Meeting of the American Association of Dental Schools. By R. S. VINSANT, D.D.S., Secretary-treasurer. 1930: pp. 411; 7 x 4 in.; no illustrations. American Association of Dental Schools, Memphis, Tenn.

Higher education in America. Edited by RAYMOND A. KENT, President of the University of Louisville, with twenty-two collaborators. (Chapter V: *The school of dentistry*, by WILLIAM J. GIES.) 1930: pp. 689; 6¾ x 4 in.; 32 illustrations; $4.00. Ginn and Company, Boston, Mass.

Prosthetic dentistry. An encyclopedia of full and partial denture prosthesis. By IRA GOODSELL NICHOLS, D.D.S., Lieutenant Commander, DC-V(S), USNR, Special Lecturer and Instructor in Dental Prosthesis, Dental Department, U. S. Naval Medical School, Washington, D. C., with collaboration of forty-five authorities. 1930: pp. 687; 8 x 5 in.; 830 illustrations; $12.50. C. V. Mosby Co., St. Louis, Mo.

Personal and community health. By CLAIR ELSMERE TURNER, M.A., Dr. P.H., Professor of Biology and Public Health, Massachusetts Institute of Technology; formerly Associate Professor of Hygiene, Tufts College Medical and Dental Schools. *Third edition.* 1930: pp. 443; 6½ x 3¾ in.; 62 illustrations; $2.75. C. V. Mosby Co., St. Louis, Mo.

Dietetics and nutrition. By MAUDE A. PERRY, B.S., formerly Director of Dietetics: Michael Reese Hospital, Chicago, Ill., and Montreal General Hospital, Montreal, Can. 1930: pp. 332; 6 x 3¾ in.; no illustrations; $2.50. C. V. Mosby Co., St. Louis, Mo.

Special cavity preparations and casting processes for gold inlays. By HARRY ERNEST BULYEA, D.M.D., Professor of Operative Dentistry, Department of Dentistry, University of Alberta. 1930: pp. 30; 7¼ x 4¾ in.; 17 illustrations. *Bulletin Number 12*, Canadian Dental Research Foundation. Toronto, Can.

Ultra-violet rays. Their application in the practice of dentistry. By ROBERT J. READE, M.A., M.D., C.M., D.D.S. 1929: pp. 31; 7¼ x 4¾ in.; 6 illustrations. *Bulletin Number 13*, Canadian Dental Research Foundation, Toronto, Can.

Annual report of investigations by the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association. *Vol. XVIII;* 22 assembled reprints; 1929. American Medical Association, Chicago, Ill.

Annals of the Pickett-Thomson Research Laboratory. Vol. V. The pathogenic streptococci; their rôle in human and animal disease (continued). Three parts on the rôle of the streptococci: in oral and dental sepsis; in tonsilitis and pharyngitis; and in puerperal sepsis and septic abortion. By DAVID THOMSON, O.B.E., M.B., Ch.B. (Edin.), D.P.H. (Camb.), Hon. Director, Pickett-Thomson Research Laboratory, St. Paul's Hospital, London; and ROBERT THOMSON, M.B., Ch.B. (Edin.), Pathologist to the Pickett-Thomson Research Laboratory. 1929: pp. 484; 8¾ x 6 in.; 49 illustrations; $10.00. Baillière, Tindall and Cox, London, England.

The physiology of oral hygiene and recent research, with special reference to accessory food factors and the incidence of dental caries. By J. SIM WALLACE, D.Sc., M.D., L.D.S., Lecturer on Preventive Dentistry, King's College Hospital; formerly Dental Surgeon and Lecturer on Dental Surgery and Pathology, London Hospital. *Second edition.* 1929: pp. 228; 6¼ x 3½ in.; seven illustrations. Baillière, Tindall and Cox, 7 and 8, Henrietta St., Covent Garden, London, England.

An outline of oral surgery for the general dental practitioner. By JAMES L. ZEMSKY, D.D.S., Attending Oral Surgeon to the Midtown Hospital, New York. 1930: pp. 171; 6 ¾ x 4 in.; 116 illustrations; $4.50. Physicians and Surgeons Book Co., Brooklyn, N. Y.

Oral diseases: a practical treatise offering diagnostic and therapeutic aid to the practitioner of medicine and dentistry. By JAMES L. ZEMSKY, D.D.S., Attending Oral Surgeon to the Midtown Hospital, New York. 1930: pp. 402; 6¾ x 4 in.; 414 illustrations; $8.00. Physicians and Surgeons Book Co., Brooklyn, N. Y.

Those teeth of yours. By J. MENZIES CAMPBELL, L.D.S., D.D.S., F.R.S.E., F.I.C.D., Fellow of the Royal Society of Medicine. 1929: pp. 141; 5½ x 3½ in.; 17 illustrations; 3/6 net. William Heinemann, Ltd., 99, Great Russell St., London, W.C.I., England.

Infant nutrition. By WILLIAM MCKIM MARRIOTT, B.S., M.D., Professor of Pediatrics, School of Medicine, Washington University. 1930: pp. 375; 7¼ x 4¾ in.; 55 illustrations; $5.50. C. V. Mosby Company, St. Louis, Mo.

Rickets including osteomalacia and tetany. By ALFRED F. HESS, M.D., Clinical Professor of Pediatrics, University and Bellevue Hospital Medical College, New York City. 1929: pp. 485; 7¼ x 4 in.; 52 illustrations; $5.50. Lea and Febiger, Philadelphia, Pa.

The properties and uses of drugs. By HENRY H. RUSBY, Ph.M., M.D., D.Sc., Dean and Professor of Materia Medica, College of Pharmacy, Columbia University; A. RICHARD BLISS, JR., A.M., Phar. D., M.D., Dean of the School of Pharmacy, and Chief of the Division of Pharmacology and Materia Medica, Colleges of Medicine and Dentistry and School of Pharmacy, University of Tennessee; and CHARLES W. BALLARD, A.M., Phar. D., Ph.D., Associate Professor of Materia Medica and Director of the Microscopical Laboratory, College of Pharmacy, Columbia University. 1930: pp. 823; 7¼ x 4¾ in.; 38 illustrations; $6.50. P. Blakiston's Son and Co., Philadelphia, Pa.

American illustrated medical dictionary. A complete dictionary of the terms used in medicine, surgery, dentistry, pharmacy, chemistry, nursing, veterinary science, biology, medical biography, etc. By W. A. NEWMAN DORLAND, A.M., M.D., F.A.C.S., member of the Committee on Nomenclature and Classification of Diseases, American Medical Association, and Editor of the "American Pocket Medical Dictionary"; with the collaboration of E. C. L. MILLER, M.D., Professor of Bacteriology and Biochemistry, Medical College of Virginia. *Fifteenth edition.* 1929: pp. 1427; 8¼ x 4¾ in.; 525 illustrations; flexible binding—plain, $7.00; thumb index, $7.50. W. B. Saunders Company, Philadelphia and London.

Fundamentals of pathology. By JOSEPH SCHROFF, B.S., M.D., D.D.S., Associate Professor of Oral Pathology, School of Dental and Oral Surgery, Columbia University. 1929: pp. 109; 7¼ x 4¾ in.; 40 illustrations; $2.50. William Albert Broder, New York City.

Outline of bacteriology. By HENRY A. BARTELS, B.S., D.D.S., Lecturer on Bacteriology, School of Oral Hygiene, Columbia University. 1929: pp. 128; 6¾ x 4 in.; 47 illustrations; $2.00.

PRELIMINARY COMMENT ON THE ORAL HYGIENE COMMITTEE'S CRITICISM OF THE AMERICAN CHILD HEALTH ASSOCIATION'S RESEARCH MONOGRAPH NUMBER III, ENTITLED "PUBLIC HEALTH ASPECTS OF DENTAL DECAY IN CHILDREN"[1]

GEORGE T. PALMER, Dr. P. H.

Director, Division of Research, American Child Health Association, New York City

The preprint of the Oral Hygiene Committee's lengthy criticism first came to our attention on September 23, 1930.[2] Other demands prevent our preparing a statement by October 18 in time for the December number.[3] Adequate treatment of this matter is merely postponed, however, till a later issue. We do wish to register immediately our concern that so much of our intent was misunderstood, and that so much of our presentation has led to miscomprehension. Much of the criticism by the Oral Hygiene Committee is out of focus. It is not aimed at true issues which exist, but rather at interpretations which do not appear in our text, but which are implied to it.

We hope for a true definition of the issues so that we may see where the data are inadequate and consequently where more research is needed. But first our true position must be clearly understood. As we wish to avoid adding any further confusion we shall make a very careful attempt in the near future to prepare answers to the errors of interpretation appearing in the Oral Hygiene Committee's report.

[1] Oral Hygiene Committee of Greater New York: "School Health Research Monograph Number III, of the American Child Health Association, entitled 'Public health aspects of dental decay in children'—An analysis, a protest, and a correction," *Journal of Dental Research*, 1930, x, p. 497; October.

[2] A preprint was forwarded to the author of Monograph III, by the *Journal of Dental Research*, on the day of its receipt from the printer's office on September 22, 1930—the earliest opportunity to do so.—[*Ed.*]

[3] A preprint of this preliminary reply was presented, by the *Journal of Dental Research*, to the Oral Hygiene Committee at its monthly meeting on November 18, 1930.—[*Ed.*]

At this time we wish to clear up, if possible, the most obvious confusion in the minds of the Committee, namely, the failure to distinguish between the publication of research findings and the announcement of an Association policy.

The American Child Health Association has been engaged for a number of years in an effort to help increase and make more effective the various public and private organized activities carried on over the country to safeguard and improve the health of children. Our Association considers that dental care is an important part of the health care of the child. The best interests of our Association lie in assuring, so far as it can, the adoption of wise policies in the administration of dental work in the public health field. In this respect we have common objectives. The large part which promotion activities have played in the program of our Association in the past may have obscured the fact that Monograph III is published as a strictly technical presentation of the findings of a research and not as a recommendation of public health policy.

We believe that the public health campaign should in increasing measure call to its aid research methods to improve the quality of judgments and opinions, and to test the results of activities that evolve from these judgments. To that end we have encouraged research and have been enabled ourselves to conduct research within the field of school health administration. Research Monograph II on "*Physical Measures of Growth and Nutrition*" contributed data which led to a new point of view in the practice of weighing and measuring school children. Guided by these findings the Association was led to a change in attitude and policy with respect to these practices, and this policy was announced in an Association Bulletin. So, too, in Monograph III we have expressed interpretations only of the dental materials as gathered. "*Public Health Aspects of Dental Decay in Children*" deals with the research aspects of public dental service. We shall hope in the future to express an Association attitude on dental service in the schools in its administrative aspects, and shall then draw upon these and other pertinent materials in formulating this attitude.

As with any product of research, the publication of the data and conclusions was for the purpose of obtaining as wide as possible criti-

cism of the research. As indicated in the first paragraph of the Monograph, we are concerned with the derivation of a means of evaluating the degree of health of the teeth in groups of children. Such a group measure is needed in judging the results of various school dental programs. The limitation of the data obtained in connection with the development of a group measure therefore accounts for the limitation of the conclusions to the immediate data at hand. We regret that such a limitation of the conclusions has been interpreted as of "destructive intent." If the Association staff had been consulted before the publication of the Oral Hygiene Committee's report, such a misunderstanding of "destructive intent" could easily have been corrected, as could have many of the other misinterpretations apparent in the Committee's report.

No one disputes the fact that further information having a bearing on the causes of dental decay in children would be welcomed. We were assured of this on all sides in our early consultations with dentists. Our Monograph is offered as a contribution in this direction. So far as we have been able to discover in a preliminary reading, nothing has been brought forward in the Oral Hygiene Committee's report that would cause us to alter, except in the way of amplification and clarification, the interpretation originally made in the Monograph. And yet we stand ready to modify our opinions when evidence of a more far-reaching character convinces us that our techniques were inadequate or our sampling was not sufficiently representative for school children of the ages covered in our study. In the meantime, in so far as other work will permit, we shall be very glad to discuss points in the Monograph that are not clear with those who would care to visit our offices. At such times, also, we shall be happy to make our files accessible and display original data and explain methods followed throughout our analysis.

370 Seventh Avenue,
New York City

A REVIEW OF DENTAL LITERATURE ON PITS AND FISSURES[1]

THADDEUS P. HYATT, D.D.S., F.A.C.D.

Professor of Preventive Dentistry, New York University Dental College, and Assistant Medical Director, Metropolitan Life Insurance Company, New York City

It is interesting to turn back the pages of dental history, and to become acquainted with the views of early writers, on the subject of enamel defects and their relation to caries. While this review is not a complete study of all dental literature, the few quotations given will no doubt be of interest to many. We shall find that the filling of pits and fissures, even before decay can be detected, has long been advocated. It is probable that the slowness in the adoption of this sound and scientific principle was due to the fact that measures for the prevention of diseases were also slow in attaining their present recognition. During the intervening years more attention and thought have been directed to the restorative and reparative phases of our work than to the possibilities of *preventing decay* by *operative* measures. Those who now oppose the idea of treating or filling pre-carious pits and fissures, believing that it represents a new theory, should have more confidence in this operative measure when they learn how long it has been advocated *and by whom.* It is very interesting to note that men who have carefully studied the locations where carious cavities most frequently appear became convinced that the shape or form of the tooth *at this point* had much to do with the liability or tendency of this place to become decayed.[2]

[1] Understanding that Dr. Hyatt had for several years been reviewing dental literature on pits and fissures, and much interest having lately been shown in this subject, Dr. Hyatt was invited to present this paper before the Oral Hygiene Committee of Greater New York, which he did at the monthly meeting at the Great Northern Hotel, New York City, October 21, 1930.—[*Ed.*]

[2] See the protest of the Oral Hygiene Committee of Greater New York on the disregard of this matter in School Health Research Monograph Number III, of the American Child Health Association, entitled "Public health aspects of dental decay in children." *Journal of Dental Research*, 1930, x, pp. 513 and 527.—[*Ed.*]

Throughout this review I shall freely italicize the parts that seem to be of particular significance in view of present day discussions. Unless otherwise stated all italicized parts were not italicized in the original articles. Most of the quotations are not continuous, only the most pertinent parts having been selected; but no qualifying remarks have been omitted.

It will, I think, surprise many to read that, in 1835, William Robertson, in his "Practical Treatise of the Diseases of the Teeth," called attention to the fact that it is the *shape* or *form* of the tooth which *increases the liability* of the tooth to decay. He wrote in part as follows:

"I propose to show in the subsequent remarks, that the *predisposition* to caries, and also the nature of the causes which excite it, are to be *discovered* in the *external form and configuration* (p. 17).

"I shall now proceed to establish, by passing successively under review the several divisions of the teeth in the order of their liability to decay; and consequently the first that claim our attention are the molars (p. 17).

"The molar teeth are much larger than any of the other teeth, their grinding surfaces are broad, and present an irregular cavity bounded by an elevated and uneven ridge. This surface is often intersected with numerous smaller projections or ridges running transversely and in various directions, and corresponding with an equal number of depressions, which constitute so many smaller cavities or deep pits; occasionally a fissure extends across the ridge of the masticating surface, and forms a cavity in the side of the teeth; in other cases the masticating surface presents three or four prominences around a deep pit in the centre. These indentations are in size, depth, and number infinitely variable, so much so that we rarely meet with two molar teeth exactly alike, *with the exception of the corresponding teeth of the same jaw, in which we always find a great similarity of structure.*[3]

"The *liability* to caries in the class of teeth now under our notice (which, as before asserted, are of all the teeth most frequently affected with this disease) will be found exactly to coincide with the irregularity of their surfaces as just described, and particularly with the depth of their indentations. Accordingly the *masticating surfaces in which the deepest depressions* are met with, are in the majority of cases the seat of decay, and the *first* to be affected with it; and the part of these surfaces in which the disease is *always found to originate* is the *bottom of one or more of the deepest indentations* (pp. 18–19).

"It has been shown that the grinding teeth are most liable to caries in consequence of their deeply-indented surfaces. In early life, when the teeth appear through the gums, the prominences upon their grinding surfaces are higher, and their depressions consequently deeper, than at a more advanced period; *and should the formation of these indentations be calculated to retain particles of food*, the operation of decay would immediately commence; and the loss of these teeth in early life would be the result. *On the other hand, it may be safely presumed that the surfaces of such teeth as remain sound to the advanced period alluded* to by *Mr. Hunter, never were deeply indented, or they would not have escaped decay;* but having escaped it, they are, in every succeeding stage of life, less and less disposed to the effects of this disease; for, we generally find that, at the age of fifty, the surfaces of the teeth are worn down, and become smooth from a long course of mastication, consequently the *cause* of *liability* to decay in this situation (*which of all others is the most liable in youth*) is entirely removed; and when decay does occur, it is principally confined to the sides of the teeth, and is occasioned by a lodgment of food in the interstices produced by a receding of the gums (pp. 29–30).

"When decay commences upon the surface of a grinding tooth the disease is confined to one or more of its deepest indentations, and may be discovered by a brown discoloration of the part, which discoloration is produced by a chemical action of the food lodging in that situation; and as this action proceeds and increases, the bottom of the indentation becomes black and corroded, and soon afterwards (by the application of a pointed instrument) a small opening may be discovered through the enamel. When the putrid substance has been admitted through this orifice to the softer bone within the enamel, decomposition in this situation proceeds with much *greater rapidity*, whereas the *opening through the enamel increases but little*, its texture being better able to resist the effects of the chemical action, on account of its greater density; but when decay has proceeded so far as to make an excavation, by the destruction of a considerable portion of the bone under the enamel, the support being removed, the enamel suddenly breaks in during mastication, and a cavity is laid open *which, till this period*, was not suspected, in consequence of the *smallness of the orifice*, and the destruction of the tooth, up to this state of the disease, being unaccompanied with pain" (pp. 38–40).

In 1853 Alfred Barron Jones published a book entitled "Observations on the Diseases and Loss of the Teeth," containing the following comment (pp. 10–12):

"The corresponding teeth on opposite sides of the jaw are identical in structure,[3] developed simultaneously, and consequently under exactly the same conditions of the system. They make their appearance externally at about the same time, and are then subjected to the same influences; I think, therefore, that in the case of the teeth, at least, the symmetry of disease may be accounted for by the perfect correspondence of all other circumstances connected with them. It follows as a natural consequence, that what produces decay in one tooth, also tends to produce it in its fellow. This is a matter of observation. The importance of paying due attention to the fact, that the teeth decay in pairs, is obvious in the treatment of this disease.[4]

"The kind of teeth most frequently the subjects of decay are those of a transparent white color. The yellow or straw colored teeth though wanting in the beautiful brilliancy which distinguishes the former, are by far the most durable, the transparent appearance being too often due to defective development."

In 1870 James Truman, published an article on fissures, in the January number of *Dental Cosmos*, from which the following is quoted:

"Whether the filling of teeth be regarded as a subject already barren of interest, or a field so thoroughly explored that no new result can be obtained, or still a theme open to further explorations, it cannot be denied that much more needs to be said to change *bad practices* if not *bad theories*. It is certainly astonishing that the progress in some directions has not kept pace with the great advances made in others. In this category may be placed the ***knowledge and treatment of those depressions in teeth technically termed fissures.*** As the proper definition of this word embraces all chasms made by separation of parts, or any narrow furrow or depression left normally in the structure, it is eminently proper to class all lines of decay met with on teeth under this comprehensive heading.[5]

"It may be regarded as an axiom in operative dentistry that all depressions, however produced, *predispose* to caries.

"If the rough surface be subjected to the constant natural cleansing

[3] Also formation or shape.—[T. P. H.]

[4] See the Oral Hygiene Committee's emphasis on this view in its discussion of the study referred to in footnote 2: *Journal of Dental Research*, 1930, x, pp. 512–14.—[*Ed.*]

[5] See the Oral Hygiene Committee's emphasis on the desirability of early treatment of all pits and fissures as a comprehensive preventive measure: *Journal of Dental Research*, 1930, x, pp. 507–9.—[*Ed.*]

process of mastication, the result may not be injurious but in the *treatment of teeth nothing should be left to the uncertainty of chance.*[5]

"If then so short a time is required to work out such serious results, it *cannot be a subject for speculation* how soon the same deposition will produce the destruction of the tissue where the *defect is more clearly pronounced.*

"If, then, we may assume that *every defective surface* will afford a nursery for the growth of germs tending to the destruction of the tissue, does it not become a matter of *grave importance that all such defects should receive prompt and careful attention at the hands of the conscientious practitioner?*

"*I regard it as positive malpractice to allow any fissure to remain unfilled where the slightest evidence of softening of enamel exists, and am satisfied that this cannot be successfully refuted.*[5]

"The assumption that time will so enlarge the defect as to render its treatment a matter of far less difficulty, but increases the *seriousness of the evil and the culpability of the operator.*

"If it be admitted that teeth may be left for further decay, a door is left open for an *amount of malpractice too serious to contemplate* without some feeling of *indignant remonstrance.*

"If there be *any doubt,* give the *tooth* the *benefit* of the doubt, and *fill the fissure. With this treatment invariably adopted, there is but slight opportunity for further decay.*"[5]

While Dr. Truman's language is strong, I doubt if it is one bit too emphatic. We must realize how very simple is the operation. We must also realize how very serious are the consequences when this simple operation is not done. The filling of pre-carious pits and fissures is so simple that, in New Zealand, young women are or will be taught to do this for the deciduous teeth and for the first permanent molars. When the child is twelve years old he will then go to a dentist.[6]

In 1871, J. H. McQuillan, M.D., D.D.S., published an article in *Dental Cosmos,* page 300, entitled: "Microscopical Fissures in the Masticating Surface of Molars and Bicuspids." In part he wrote:

"In the accompanying illustration it will be observed that a minute fissure, invisible to the naked eye in the section, extends from the bottom of the sulcus on the grinding surface of the tooth, through the enamel, almost to the dentine and enlarging at the lower part into an oval cavity. This is

[6] Reliable private information.—[T. P. H.]

entirely the result of *defective formation*, the enamel prisms having *failed to coalesce at that point*, and thus a condition is presented *favorable* to the *retention* of fluids and semi-solids, which undergoing decomposition would speedily destroy the thin septum of enamel covering the dentine.

"In a paper read before the American Dental Association at the meeting held in Boston, August 1866, giving the results of a personal examination of the interglobular spaces, I remarked: 'As evidence of the practical bearings of these investigations, it may be well to direct attention to the fact that the existence of the spaces in teeth which have completed their growth must be regarded as an abnormal condition, predisposing such teeth to decay, and that when either by mechanical action, as by a fall, or blow, or by the penetration of external caries, such spaces are reached, the disease here would run riot; hence the importance of care on the part of patients and operators to have the most minute cavities filled; for though reached only through a microscopical opening, the result would be the same, while, *if protected from the action of external influences or the exciting causes of decay, this predisposition might remain dormant for a life time*, as is sometimes the case with other diseases.'

"It is to be hoped that the illustration offered will teach a valuable lesson to those who have been in the habit of dismissing their patients with the statement that *'there are some small cavities in the teeth which can be left without disadvantage until another time.'*"

I am reliably informed that *even today* many dentists, in different parts of this country as well as in Europe, tell their patients that small openings in the enamel may be left until they become bigger. In view of what Dr. Truman has said, the truth of which is self-evident, it is hoped that before long our profession, becoming jealous of its reputation and the integrity of its members, will take drastic measures to prevent this, even to the extent of invoking the assistance of the law.

In 1871, Robert Arthur, M.D., D.D.S., published a book entitled: "Treatment and Prevention of Decay of the Teeth," from which the following is copied:

"We have, then, all the elements to account for the decomposition of the teeth. But if they are constantly in contact with a fluid more or less acid in character, the natural result to be looked for would be their general decomposition in a manner similar to what occurs when an extracted tooth is immersed in dilute acid. This, however, does not take place. Decay of the teeth is not a uniform decomposition of their surfaces. The destructive process is seen to begin at *a particular spot*, sometimes more than one, from which it extends in every direction, but more rapidly toward the nerve.

"Whatever may be the source of the agent causing decay, the disorder is always *observed to commence at points which favor the lodgment and retention of particles of food*, or the secretions of the mouth.[7]

"Whenever, therefore, a *defect* in the continuity of the enamel of a tooth exists, *decay is sure to occur. To this rule there are very rare exceptions. It is not necessary that the defect should extend through the enamel. Decay commences at the defective point.*[8]

"*The perfectly-formed portion of the enamel* on the grinding surfaces of the teeth surrounding the defective places noticed, *is not liable to decay*, as it is *kept cleansed* by the act of mastication itself.

"It has been explained that decay does not usually attack the teeth except at such places as favor the lodgment and retention of particles of food or the secretions of the mouth. On the broad grinding surfaces of the molar and bicuspid teeth, or the cutting edges of the incisor teeth, *decay would never take place, provided the enamel on these surfaces were well and smoothly formed.* Nor does decay, except in very rare instances, attack the smooth surfaces of any of the teeth toward the lips, cheeks or tongue. The act of mastication, the almost constant movements of the tongue and lips, and the copious flow of the saliva, tend to keep the more prominent parts of these surfaces free from extraneous deposits of any kind. But the enamel of the grinding surfaces of the teeth, as has been explained, is very rarely perfectly formed. Defective places remain in some parts of this covering of the dentine almost invariably. *At these places*, as already explained, decay is very nearly certain to occur.

"*Now, it is obvious that if these defective places could be obliterated, by being filled up with some substance capable of resisting the action* of such decomposing agents as are present in the mouth, *decay* could not occur."

In 1899, R. R. Andrews presented, in *Dental Cosmos*, page 719, a review of this subject in a paper on "The Pits and Fissures of the Enamel," from which we quote as follows:

"Fox in 1803 described fissures. He believed that the teeth acquired this disposition to decay from some want of healthy action during their formation, which was proven by their becoming decayed in pairs.

"In 1835 William Robertson devoted much attention to pits and fissures and stated that *surfaces having pits and fissures are far more liable to decay.* He recognized that it [decay] *never occurred on clean* and smooth surfaces,

[7] See the Oral Hygiene Committee's emphasis on the influence of local factors in decay: *Journal of Dental Research*, 1930, x, pp. 530–31.—[*Ed.*]

but that in all instances the attack was made at *such points as collect and retain the food until fermentation takes place*, as in the interstices between the teeth and in pits and fissures in the enamel. Robertson also believed that pits and fissures were the principal cause for the destruction of the teeth. Robertson also believed that the liability in different individuals to decay of the teeth *was in proportion to the form and depth of these fissures.* Therefore when a tooth has appeared above the gum we can *readily ascertain whether it is or it is not predisposed to decay* by examining the structure of the enamel.[5]

"Kelly (1843) tells us that decay commences beneath a fissure . . . . the disease burrows for a longer or shorter time so far within the crown of the tooth as *to give little or no external indication* of its true condition.

"Those who argue that the constitution has but little to do with the teeth suppose that a fissure can always be found over the point where this variety of decay occurs, and hence the only exciting causes are outward and accidental. Allowing this defect to exist, it must be admitted, for it is proof itself, that the constitutional powers were originally unequal to the perfect organization of the tooth, and, consequently its powers of resisting destructive agents are below the natural standard, which in the teeth are, at best, lower than in other parts of the system. *It is obvious, then, that when any modification of the general health, or any local causes, dispose the teeth to decay, it will be seated where they are least protected on their surfaces in the fissures.*[8]

"Tomes says: 'From the natural depressions which separate the cusps of molars, minute but deep fissures may be extended through the enamel to within a short distance of the dentine, *and they may become larger as they recede from the surface of the tooth.*'

"He claims that in the *foremost rank*, as a *predisposing* cause to decay, must be placed these *deep but minute fissures* found upon the masticating surface of the molars and bicuspids.

[8] This paragraph is very significant, and should receive careful thought and attention. A tooth having a pit or fissure is sufficient proof "that the constitutional powers were originally unequal to the perfect organization of the tooth." This being true, it logically follows that children having teeth with pits or fissures *are particularly susceptible to decay.* This should be a most significant indication that greater care and earlier attention must be given to these cases, if we wish to attain any success in the preservation of the teeth. Not only must we give attention at once to every enamel defect, but we must also give strict attention to diet. The greatest emphasis must be placed on the proper home care of the mouth. Parents should be most profoundly impressed with the absolute necessity of giving the greatest care to the general health of the child. They must be shown that only in this way can the child be spared all the trouble, pain, and probable loss of many teeth which follow when *decay is allowed to start.*—[T. P. H.]

"Saltar (1875) taught that *the defects in the enamel* between the cusps of molars and bicuspids are common and fruitful of destructive diseases.

"Now, these disturbances, when they occur, ought necessarily, owing to the law which governs them, to be produced simultaneously, and in the same degree, in all the teeth which are at the same moment in the process of dentification. This is in fact what happens and here is found the explanation of identical congenital lesions upon homologous teeth, and consequently of decay which has the same relative position. It is *not then surprising* to see two molars, for example, or two incisors on opposite sides of the same jaw, *presenting the same fissure*, the *same crevice*, the same congenital cavity, and in consequence one position, one progress, *and one identical form of two parallel cases of decay.*"[4]

Dr. A. Biro read, before the Royal Hungarian Society of Physicians, in Budapest, a paper published in the (Dental) *Items of Interest* for September, 1898, "On the Predisposing Causes of Caries." Following Dr. Biro's paper is a discussion by Dr. J. Leon Williams. Dr. Biro took the position that "the predisposing *causes* of dental caries in the tooth are: "(1) Shape or form; (2) density or structure; (3) reaction or vitality." Dr. Biro wrote: "In the first category belong the various defects of the enamel as well as the imperfect form and position of tooth crowns, which are accompanied by irregular interdental spaces. Since the origin of caries has been discovered these are known to be predisposing causes, inasmuch as they give opportunities for remains of food to lodge and accumulate." Dr. Williams very correctly took exception to Dr. Biro's use of the word "causes" as not being logical. It is the shape or form of the tooth that constitutes the factor which increases or lessens the liability of these teeth to decay. It is the shape or form of a surface, and also the location of the surface, that largely determine the liability of this surface to decay. These pits and fissures are only contributory factors, which enable certain *conditions* to exist. Were they "causes" would other factors be needed, such as food debris and bacteria? Is it not difficult to claim any one factor as being "*the* cause?" If there were no food debris there would be no bacteria. If there were no bacteria, there would be no acid of sufficient strength to break down the enamel. If there were no undisturbed retention of food debris and bacteria, with warmth and moisture, there would be no decay. Constitutional

conditions and diet are also important contributory factors. Therefore there are many factors, the combination of which comprise that complex something which we call *the cause.*

In 1899, C. N. Johnson, now the Editor of the *Journal of the American Dental Association*, contributed an article on "A Few Considerations in Filling Teeth," in *Dental Cosmos* for January. Under the heading, "Occlusal Cavities in Bicuspids and Molars," he wrote:

"These cavities are usually the result of structural imperfections in the tooth by which the developing islands of calcification, beginning at the tips of the cusps, have failed to properly unite on approaching each other, *leaving a leak* for *the ingress of foreign matter.*

"An important distinction between caries occurring upon these surfaces and that of other surfaces already considered *is due to the fact that upon occlusal surfaces decay seldom occurs except as the result of defects in the enamel*, while on the others it is often found beginning upon perfectly formed enamel. *The reason for this is that the friction of mastication very largely prevents the possibility of decay upon the occlusal surfaces, except as the agent of caries is harbored in some crevice or fissure where the cleaning process of mastication cannot reach.* On this account extension for prevention is seldom indicated on occlusal surfaces unless the drilling out of all fissures running from the cavity may be so interpreted.

"This problem of the treatment of fissures is one indissolubly linked with the management of these occlusal cavities. *Many operators do not seem to consider it necessary to drill out fissures unless actual decay has begun in them.* They overlook two things: the difficulty of making a good margin to the filling at the intersection of a fissure, and the real nature of the imperfection that a fissure represents. If an operator has any doubt as to the necessity for drilling out all fissures extending from a cavity under preparation, let him make a microscopical examination of sections of teeth cut crosswise of a fissure, and he will no longer hesitate. In many of these cases where the orifice of the fissure is so narrow as scarcely to admit the finest exploring instrument, the microscope will show a decided imperfection reaching entirely through the enamel. . . . *This kind of a break in the enamel-surface is a serious menace to the tooth*, and *no operator* is doing *his duty* by the patient when he *leaves* such a *defect* in connection with his work. It is the minutiae which count in dental practice, *and microscopic conditions must not be ignored.*"[5]

Dr. Johnson is almost as strong as Dr. Truman when he claims that no dentist is doing his duty when he neglects these fissures *even though*

no decay is seen.[8] In the fourth edition of "Principles and Practice of Filling Teeth," of which Dr. Johnson is Editor (1918), he states on page 26:

"Saliva in the mouth cannot become sufficiently acid to penetrate the teeth in the way we find decay manifests in most cases. If it were so sharply acid the soft tissues could not tolerate it, and, besides, if it was the saliva which did the work we should find the entire exposed surfaces of the teeth melted down instead of being penetrated *at certain points*."

Dr. Johnson then goes on to explain about the gelatinous film under which the microörganisms produce decay, and states:

"But the film may be broken up and detached by any appreciable amount of friction, such, for instance, as the friction of food in mastication where that process is exercised to its fullest functional activity. Accordingly, we may look for the greatest ravages of the teeth not subject to the friction of food, as the proximal surfaces *or in sheltered localities formed by developmental defects as in fissures or pits where the micro-organisms may work unmolested*, and this phenomenon is amply demonstrated in clinical observation.[9]

"*An operator owes it to his patient not to overlook the slightest* defect.

"As has already been stated a fissure is invariably a defect in the tooth whereby two islands of calcification have failed to coalesce in its development leaving a break in the continuity of the enamel at that point *and a crevice for the ingress of deleterious matter.*"

In his day no man gave greater attention to the value and importance of statistical investigation of dental caries than did G. V. Black. *Dental Cosmos*, in December, 1909, at page 1390, published a paper by Dr. Black that had been presented before the Pennsylvania State Dental Society in Pittsburgh, on June 20, 1909. Dr. Black said in part:

"*Acid produced and dissolved in the saliva has no effect in producing caries.* Acid saliva is of no consequence whatever in relation to dental caries. Persons immune to caries have acid saliva just as persistently as those susceptible to caries.

"Caries of dentin is hidden away within the tooth, and unless the opening

[9] We may truthfully add: *and proven by statistical findings.*—[T. P. H.]

into the decayed area is broad, is beyond outside influences. It is true that the organisms causing it must receive sugar from the saliva, but this is obtained by osmosis, or through the process of dialysis, the softened dentin acting as the dialyzing medium. Hence for caries to progress in dentin there must be an opening to the surface, *but the progress is more certain if the opening is small.*

"We find that caries always has its beginning in the enamel in the out-of-the-way places upon the teeth, such as in the pits and fissures of the occlusal surfaces where colonies of microörganisms may grow, covered in and protected by food debris or gummy material not soluble in the saliva.

"*Caries never attacks the enamel in the open, where the surface of the tooth is freely cleaned,* exposed to washings by the saliva and to the crush of food over the surfaces in chewing. Therefore decay of the enamel beginning on the surface is *strictly limited to certain areas of tooth-surfaces,* and superficially cannot spread beyond those areas in any case where the person is making normal use of the teeth in chewing food. *This is the cause of the limitation of the beginning of caries to these limited areas.* These areas may, upon examination, be mapped out *very certainly in any mouth,* and we shall find that practically all beginnings of caries on the surface of the enamel will be confined to them. Therefore the beginning and the superficial spreading of decay of the enamel is subject to these *purely local conditions even in the most susceptible persons.*[7]

"In occlusal decay occurring in pits, *there is no spreading on the surface of the enamel.* The decay, beginning in the depths of the pits, penetrates to the dentin and at once spreads in that tissue, undermining the enamel, which slowly crumbles from backward decay. *Therefore the treatment of these cavities is the simplest of all occurring in the mouth.*"

In 1910, in an interesting article on the "Preparation of Cavities for Fillings," by Dr. A. E. Webster, in "A Text-Book of Operative Dentistry," edited by Dr. C. N. Johnson, the following appeared:

"Pit and fissure cavities are the result of defects in the enamel covering of the tooth, and are to be found in the occlusal surfaces of biscuspids and molars, lingual surfaces of incisors and occasionally in the occlusal third of the buccal surfaces of molars. The enamel begins to calcify at several points, the central incisor at three points, the bicuspids and molars at the tips of the cusps. As the calcific matter is deposited the different lobes of enamel should join between the cusps, *but this does not always happen, and as a consequence there is left a fissure which is a defect in the continuity of the*

surface and must be distinguished from a groove which is only a depression and not a defect. The great majority of all fissures are the seat of caries sooner or later. The conditions are the most favorable for development of microörganisms.

"If it were not for the pits and fissures in teeth, caries would rarely occur in occlusal surfaces, because they would be kept clean by the mastication of food.

"If every operator had the opportunity of seeing the microörganisms of decay under a microscope, and then seeing the width of the finest fissure under the same power, there would be no doubt in his mind as to what should be done. Fissures are defects *and are always a source of weakness.*"[5]

At the Sixth International Dental Congress in London, during August 1914, H. W. P. Bennette, M.D.S. (Liv.), L.D.S. (Eng.), in a paper on "Principles of Cavity Preparation," made the following remarks:

"For convenience of description I will divide the types of cavities into three groups: (*a*) Those rendered necessary, or brought into being by imperfections in development, *such as fissure cavities or pits.* (*b*) Those which occur upon smooth surfaces. (*c*) Those which involve both of the above. In Group (*a*) I use the words, "rendered necessary by imperfections in development," wishing to imply that I do not hesitate to cut out fissures or pits, *even though they are not as yet seats of caries.*" (*Items of Interest:* 1914, page 923; December.)

Dr. M. D. Huff, of Atlanta, Ga., in a paper on "Present-Day Problems of Operative Dentistry" read to the Maryland State Dental Association in May, 1926, made the following statement (*Dental Cosmos*, lxix, p. 143):

"Operative dentistry formerly dealt with methods of removing decay from the teeth and the placing of fillings in cavities thus made, but, in the light of present knowledge, the logical time for operative procedure is when a tooth is found to be defective—before decay is present."

Dr. Samuel Goldberg of Johannesburg, South Africa, in a paper on "The Dental Arches of Identical Twins," in *Dental Cosmos* for August, 1930, wrote in part as follows (page 181):

"As regards dental decay, a study was made by Bachrach and Young in London, of the total incidence of caries in 130 pairs of identical twins and

171 pairs of fraternal twins. There was not a significant difference in the coefficient of correlation, and it was concluded that heredity did not affect the decay process as such. An attempt was made by the writer to study the distribution of decay in each individual mouth. The incidence of decay commencing on smooth surfaces was very haphazard, but that commencing in pits and fissures was rather uniform. In fact, the workings of the asymmetry mechanism could frequently be traced in the pit and fissure formation, and a number of identical twins showed decay in the corresponding teeth. *Hence it is concluded that heredity affects dental decay only inasmuch as it controls the shape of a tooth and its pits and fissures and its position in the dental arch,* but has no influence over the chemical composition or microscopic structure that will encourage or prevent decay. There is merely a predisposing localizing factor for dental decay of genetic origin. These findings augur well for the future control and prevention of dental decay, and agree in the main with the conclusions drawn since 1900 from the work of others who have approached the problem through microscopic, chemical, and bacteriological channels."[10]

H. P. Sutcliff, L.D.S., R.C.S., England, has long been associated with children's dentistry in Derbyshire, England. In the *British Dental Journal,* January 1, 1930, page 12, he states:

"It is a general principle with our staff to fill or dress when possible all decayed deciduous teeth, *and to fill all fissures of six year molars.* This latter point may be open to argument, but I give as my reason the fact that approximately 90 per cent of first permanent molars require fillings before the child is eight years old. *I therefore regard this procedure as sound constructive work.*"

After all that has been written on this subject, after all the microscopic research work that has been done—after all the data that have been secured from many widely different sources showing where the largest number of carious cavities actually occur, it seems strange that Mr. Sutcliffe should be obliged to admit that, in the year 1930, there are some dentists who do not believe in the early treatment of pits and fissures. Such persons remind me of the man who said two and two do not make four. Caries is by no means a product of "*accidental*

[10] See the Oral Hygiene Committee's report on this subject: *Journal of Dental Research,* 1930, x, pp. 530–31.—[*Ed.*]

combinations of circumstances," but rather a definite result brought about by *definite* combinations of circumstances.[10] These definite combinations of circumstances, as to pits and fissures, are a definite opening in the enamel; a definite protection for the influences or agencies of decay in these openings; a definite hindrance to the cleansing of such surfaces through mastication, toothbrushing, or other means. What is the definite result? Dental caries. What should we do? Promptly remove or close these enamel defects, as advocated by so many of the best men in our profession for many years. What would be the definite result? A reduction of 50 percent to 70 percent in the number of *carious* cavities.

I desire to express my thanks to Miss Lawrence, Librarian of the New York University Dental College, who rendered valuable assistance in locating articles on this subject.

MEDICO-DENTAL CASE RECORDS: NINTH REPORT[1]

A Clinico-Pathological Study

BISSELL B. PALMER, D.D.S., F.A.C.D., and MALCOLM W. CARR, D.D.S.
New York City

CONTENTS

I. Introduction 675
II. Case C-2: Myocarditis 677
General history 677
Physical examination 678
Clinical pathology (laboratory reports) 679
Diagnosis 679
Operation and treatment 679
Post-operative course 680
III. Summary and discussion 681
IV. Conclusions 683

I. Introduction

This is the third report, in this series of case records, to be devoted to the study of the relation of chronic focal infections to diseases of the heart. The first report[2] was a study of endocarditis, apparently secondary to foci of infection in both tonsils and teeth. The patient improved only slightly following the removal of the tonsils, the principal symptoms remaining until the dental infection was eradicated. The seventh report[3] was a study of rheumatic carditis and polyarthritis, a manifestation of multiple secondary lesions existing concomitantly and arising from the same primary source. The following report is a study of myocarditis, and the exacerbations that followed the removal of two infected teeth.

[1] Presented at the tenth anniversary meeting of the New York Section of the International Association for Dental Research, Columbia University Club, New York City, December 10, 1930. The previous reports were published in the *Journal of Dental Research:* (1) 1926, vi, p. 283; (2–4) 1927, vii, pp. 115, 275, 457; (5–6) 1928, viii, pp. 73, 579; (7) 1929, ix, p. 89; (8)1930, x, p. 173.

[2] Palmer and Carr: *Journal of Dental Research*, 1926, vi, p. 283.

[3] Palmer and Carr: *Ibid.*, 1928, viii, p. 73.

In this study of the relationship of focal infection to the dysfunctions of the heart, observations have been made, and data recorded, as to the reaction of the heart following the surgical eradication of primary foci of infection. The study of these reactions has included a detailed comparative study of the subjective as well as the clinical symptoms of each individual patient. Particular attention has been given to the study of the exacerbations that follow interference with these chronic areas of periapical infection. These potential areas may become dangerous foci when the bacteria or their products are suddenly liberated and absorbed into the blood, particularly when the virulence of the infection is overwhelming or the resistance of the patient is insufficient to accommodate any sudden additional load. The removal of many infected teeth at one operation may be a hazardous procedure. Many surgeons have experienced unfortunate results, before learning the fallacy of such radicalism. Not only may severe constitutional symptoms, such as fever, shock, and prostration occur, but also severe and alarming local exacerbation of the secondary disease may rapidly ensue, and fatal termination may result.

In these case reports mention has been made repeatedly of the desirability of removing at the initial operation only one or two teeth for those patients who have many infected teeth to be removed, and the potential danger of multiple extractions and the advisability of stage operations has likewise frequently been emphasized. The assumed responsibility is too great to minimize the importance of this doctrine, particularly when treating patients who have manifestations of secondary disease. Exacerbations following minor operations, however, are not always explicable in terms of virulence of infection or resistance of the patient. Inordinately severe reactions not infrequently follow the simplest operations. In some of these cases the complex problem of psychiatry is involved. In a brief study conducted by one of the authors,[4] some interesting observations were made on a number of selected patients who developed the symptoms of complete syncope and minor shock following the injection of 2 cc. of

[4] Carr, Malcolm, W.: The physiological effects of the emotion of fear upon the circulatory system. An unpublished manuscript of a preliminary report, submitted in 1923 to the Department of Physiology, School of Medicine, University of Pennsylvania, Philadelphia, Pa.

normal saline. All of these patients had previously fainted, either in the presence of the author, or gave a history of fainting following an injection of procain for a dental operation. Under similar conditions the normal saline was injected and in all the cases referred to, the pulse became weak, the blood-pressure dropped, and there was excessive pallor, dilation of the pupil, and unconsciousness. The emotions of fear and anxiety elicit very definite constitutional effects. Patients who approach a severe crisis without fear or anxiety certainly do better than those who are quite certain that only grave eventualities are to develop and face the situation with premonition, with fear and anxiety, and without hope. This has been a common bed-side observation. The following report is published because of the unusual and severe constitutional sequelae of a simple and minor operation.

II. Case C-2: Myocarditis*[5]

General History

A widow, 62 years of age, was admitted to the private pavilion of the hospital[6] on September 2, 1924, to have her infected teeth removed, although her chief complaint* had been shortness of breath* upon exertion.

*Family history.** The patient's mother died suddenly from heart disease at 71; her father, at 94 from old age. One brother died at 50, cause unknown; another, of "stomach trouble." Three brothers and one sister are living and well. The patient had four children: one died of typhoid fever; three are living and well.

*Past medical history.** She gave no history of scarlet fever, diphtheria, typhoid fever, rheumatic fever, or chorea; had measles in childhood. The patient had always been well; had four confinements, all of which were normal; surgical history, negative; used neither alcohol nor tobacco.*

*History of present illness.** For the last year the patient had been troubled with shortness of breath upon exertion. In June, after a short run, she had a severe attack of dyspnoea.* At six o'clock the next morning she had another attack of dyspnoea. There was pain.* Upon the advice of her physician, she remained at rest in bed for about a week, when she was allowed to get up for a short time. The second time she was out of bed she had a peculiar attack, which it was difficult for her to describe beyond the

[5] Words and phrases followed by an asterisk have been explained in the section on "notes and comments" of previous reports. See footnote 1.

[6] The Fifth Avenue Hospital, New York City.

fact that she felt mentally dull and was unable to give expression to thoughts. This condition continued for several days and gradually improved. Following this attack she was admitted to the hospital[6] and remained at rest in bed for three weeks. The marked cardiac irregularity with which the patient was admitted to the hospital improved with rest. She was discharged "improved," with caution to rest, not to over-exert, and to reduce weight by diet. However, this initial attack seemed to precipitate the more or less continual disturbance that has been manifest meanwhile. Since her discharge from the hospital she has felt somewhat improved. However, she has had a rapid pulse after exertion or activity, and has had to spend some time in bed following any exertion in order to recuperate. Since June, she has been under digitalis* continually. Recently the patient has complained of pain and some stiffness in the extremities. Roentgenograms show three infected teeth. The patient's second admission to the hospital was for the removal of these teeth.

PHYSICAL EXAMINATION

Examination showed a well developed and well nourished woman of middle age; she did not appear acutely or chronically ill. *Color:* good; no cyanosis;* breathing normally. *Intelligence:* above the average. *Emotions:* she was cheerful, and apparently had little anxiety or apprehension regarding the minor operation she was to undergo. *Eyes:* react normally to light* and accommodation;* pupils equal in size. *Ears and nose:* negative.

Mouth and teeth. Clinical examination.—There was a slight deposition of salivary calculus, and simple gingivitis. There were many fillings, inlays, and jacket crowns, all of which had been exceedingly well executed. Oral hygiene was good. Electrical test for vitality indicated all teeth were vital excepting the right maxillary canine, right maxillary second molar, and right mandibular second premolar. *Roentgenographic examination.*—Roentgenograms revealed moderate alveolar resorption. There were superficial subgingival pockets of infection mesial to the left mandibular second molar and right mandibular second molar. The right maxillary canine was pulpless; the root canal was well filled. There was chronic rarefying osteitis* with granuloma at the apex. The right maxillary second molar was pulpless; the root canals had been well filled. There was chronic proliferative pericementitis.* The right mandibular second premolar was pulpless, with an incompletely filled root canal and chronic proliferative pericementitis. All other teeth and surrounding bony structure appeared natural. *Recommendation.*—It was recommended that these three pulpless teeth be removed in *two stages* under local anesthesia (procain 1 per cent).

Tonsils: slightly enlarged, but not infected. *Accessory sinuses:* negative. *Sub-maxillary and deep cervical glands:* not palpable or tender. *Chest:* expansion good and apparently equal on both sides. *Lungs:* clear, no râles,* no moisture. *Heart.*—Apex beat* not visible or palpable; area of cardiac dulness enlarged* to the left; action fairly forcible but irregularly spaced beats. At the apex the first sound* was of fair muscular quality only. At the base $A_2 = P_2$;* no murmurs* elicited. Pulse:* irregular in both rate and rhythm. *Abdomen:* Abdominal wall very obese and flabby; *liver** and *spleen** not palpable, no masses* or tenderness.* *Extremities:* no edema.*

CLINICAL PATHOLOGY (LABORATORY REPORTS)

Temperature and respiration were within normal range. *Blood pressure:** systolic, 170 mm.; diastolic 90 mm. *Urinalysis** (*Sept. 2*): reaction, acid; specific gravity, 1.015; albumin, very faint trace; sugar, acetone, diacetic acid, absent. *Microscopic examination.*—Casts, moderate hyaline; red blood-cells, absent; white blood-cells, occasional; epithelial cells, moderate squamous. *Hematology** (*June 14*): hemoglobin, 100 per cent; red blood-cells, 5,500,000; white blood-cells, 6,900. *Blood chemistry** (*June 14*) (mg. per 100 cc.): urea nitrogen 10.1; creatinin 0.8; uric acid, 218; carbon-dioxide combining power, 58 per cent (volume); sugar, 105.2. *Blood complement-fixation test:** negative.

DIAGNOSIS

(a) Myocarditis,* auricular fibrillation,* cardiac insufficiency.* (b) Arteriosclerosis.* (c) Oral sepsis.

OPERATION AND TREATMENT

Medical treatment. Before operation the patient was given codeine, gr. I, and had received her usual daily administration of digitalis.

Oral surgical operation. At 2 o'clock on the afternoon of September 2, the right maxillary canine and right maxillary second molar were removed without difficulty under procain anesthesia. Five cc. of a 1 per cent procain solution containing epinephrin 1–40,000 were employed. The right mandibular second premolar was to have been removed at a subsequent operation. The pathological condition at the apex of the canine was found to be a chronic rarefying osteitis with granuloma. The second molar also showed definite evidence of infection, principally proliferative pericementitis. The sockets were lightly curetted, and the usual dressings employed.

As the patient came into the operating room, she was exceedingly nervous and showed signs of marked apprehensiveness and anxiety. She feared the injection, questioned whether the teeth should be removed, and gave expression to a subconscious feeling that grave eventualities, including death, might result. There were no objective signs to contraindicate the operation. Her pre-operative pulse, respiration and temperature were within the normal range. The importance of her then developing subconscious anxiety-syndrome was minimized, and after some persuasion she submitted, without resignation, to the injection. The operation was uneventful. The patient was returned to her room in good condition, excepting for severe anxiety and slight hysteria.

POST-OPERATIVE COURSE

Sept. 2, 3 P.M.—Patient complained of cold feet; hot-water bag placed at feet; patient still nervous. *Sept. 3, 8 A.M.*—Patient had had some rest before 1 A.M.; after 1 o'clock extreme nervousness again became manifest; given codeine gr. I by mouth, which was later vomited. Temperature 100; pulse 66 and fairly regular. Heart sounds: fair muscular quality. Enema given with good results. *12 Noon.*—Temperature 99, pulse 56, respiration 18; patient nervous and apprehensive; given digitan tab. I; later vomited twice; digitan ordered discontinued. *4 P.M.*—Condition now quite different. Pulse very rapid— rate 140 and poor quality; respiration 30. First heart sound: devoid of muscular quality; patient cold and clammy. Morphine sulphate grs. $\frac{1}{4}$, atrophine grs. 1/100 and adrenalin 0.5 cc. given hypodermicly *stat.* *7:30 P.M.*—Pulse 128, respiration 24. Nervous restlessness and apprehensiveness had abated to some degree; patient had rested for about three hours. *8 P.M.*—Patient had rested quietly from 7 to 8 P.M.; at 8 o'clock there was a definite change in the general condition, which now became similar to that at 4 P.M. Pulse 150, respiration 26. Patient given digitan amp. I hypodermicly and paraldehyde per rectum on S.O.S. for restlessness. *11:45 P.M.*—Paraldehyde had had little effect, digitan amp. I hypodermicly was given *stat.* Paraldehyde again given per rectum. Patient now again very restless. Paraldehyde expelled. Patient given morphine sulphate grs. $\frac{1}{4}$ hypodermicly. *Sept. 4, 1:30 A.M.*—Pulse weak, patient dosing; skin cold and clammy; outlook very bad. *3 A.M.*—Condition unchanged, adrenalin amp. I given hypodermicly; blankets and hot water bottle applied. *6 A.M.*—Unable to get pulse, patient unconscious; adrenalin amp. I given hypodermicly *stat.* *6:52 A.M.*—Patient ceased breathing.

III. Summary and Discussion

The patient was 62 years of age and had been troubled with shortness of breath for about a year. She gave a definite family history of cardiac disease. The past medical history was negative. Three months before her second admission to the hospital she entered, principally for rest, following her first acute attack of dyspnoea. The then existing cardiac irregularity and dyspnoea improved with rest, but the initial attack seemed to precipitate the continual subsequent disturbance. She had had digitalis continually. It seems likely that she had an attack of coronary thrombosis as her initial condition, and that the auricular fibrillation from which she suffered was due to the involvement of the conducting mechanism in the infarct. She rallied from this attack, except for the fibrillation, which continued. In the attempt to eliminate any toxic influence from the myocardium, she was admitted to the hospital for the second time for the removal of the infected teeth. At this time she showed no signs of acute illness and seemed, indeed, to be in a fair state of health. The findings of the physical examination revealed no symptoms severe enough to contraindicate the operation for the removal of the infected teeth, nor was she in any sense considered a poor risk. Certainly patients with more advanced cardiac decompensation, who actually were bad risks, have had operations more severe than this patient's and have made uneventful recoveries. Yet this patient had two (what may be considered slightly infected) teeth removed, and death resulted in a little more than thirty-six hours. Her final attack might have been due to another coronary thrombosis. Her symptoms of shock would very well go with this condition. That such a severe exacerbation might result from toxic or infectious causes alone, considering the pathological condition of the teeth,[7] seems most unusual if not unlikely.

The emotions of fear and anxiety had certainly disturbed the patient's equanimity, and it is not improbable that nervous influences were predisposing factors in the fatal termination.

In considering the functional disturbances of the various organs, frequent reference has been made to those that depend upon nervous influences. We may recall here the vagus arrhythmias and the vagus

[7] Cultures unfortunately were not taken.

alterations of heart rate, emotional syncope, nervous hyperchlorhydria and pyloric spasm, nervous glycosuria, bronchial spasm from vagus stimulation, fever following brain injuries, and the reference of pain from various organs to the surface of the body. In discussing these phenomena Hewlett[8] says:

"The activities of the internal organs are by no means so dependent upon nervous control as are the activities of the voluntary muscles. If the nerve supply to the latter is interrupted their activities cease. If it is interrupted to the former no serious result necessarily follows. The heart continues to beat when all its extrinsic nerves are severed, the arterioles ultimately regain their tone after having been isolated from their nerve supply, and other analogous relations might be cited. Nervous impulses direct and modify the activities of the internal organs, but in most instances they are not essential for a continuation of these activities.

"The study of nervous disturbances of the internal organs in patients leads to similar conclusions. In themselves, these rarely threaten life, but they play an important rôle in producing and modifying the symptoms of various ailments, and they may at times be the determining cause of a serious train of disturbances. For example, the rise of blood pressure caused by strong emotion may be the exciting cause of a cerebral hemorrhage or an anginal seizure. Prolonged nervous strain is said by some to play a rôle in producing such serious conditions as chronic arterial hypertension, diabetes mellitus and hyperthroidism.

". . . . Alterations in the visceral functions may be initiated by changes in the *higher cerebral centers*. The major emotions, fear and anger, are accompanied by changes in the cardiac activity, by changes in the blood pressure, by hyperglycemia and by a depression in the secretory and motor functions of the gastro-intestinal tract. Syncope may be due to emotion. Fever may be caused by injury to the brain and particularly to the basal ganglia. The bradycardia of meningitis or increased intracranial pressure is due to stimulation of the *vagus center in the medulla*. Medullary injury may also cause a discharge of glycogen from the liver or alterations in the respiration.

"Finally, the functions of the internal organs may be disturbed by *toxic substances* which act upon some portion of their peripheral nerve supply. It has been demonstrated that many drugs affect the visceral nervous system in this manner, and it seems not unlikely that various toxic substances

[8] Hewlett, Albion Walter: Pathological Physiology of Internal Diseases (Functional Pathology), 2nd ed.; D. Appleton and Co., New York and London.

which may develop within the body act in a similar manner. Whether such toxic or infectious causes act primarily upon the visceral nervous system, or whether they influence the parenchyma cells of various organs directly, is difficult to decide; but the involvement of function in many different organs frequently suggests an action upon the nervous structures which are common to all, rather than intoxication of the parenchymatous structures, which differ so widely, one from the other."

Hewlett's conception of systemic visceral neuroses seems logical.

IV. Conclusions

(1) The fatal exacerbation in this case might have been caused by toxic or infectious absorption and its direct effect upon the parenchyma cells of the heart or the heart muscles, or by its effect upon the visceral nervous system. It seems more likely, however, that the severe functional disturbance depended more upon the nervous influences of the symptom-complex activated by the major emotions of fear and anxiety.

(2) Functional disturbances of the heart may be exaggerated by nervous influences, of which the symptom-complex of fear-and-anxiety neurosis is an important factor.

(3) Sufficient study and *individual* consideration frequently is not given to the neurotic or psychotic patient.

(4) The removal of chronic periapical foci of infection in patients with manifestations of secondary lesions is usually an operation of choice. Therefore, operation should proceed only after a careful study of the patient's general condition, and then with extreme circumspection, and perhaps the removal of only one tooth at the initial operation.

667 Madison Avenue

ORAL INFECTIONS AND THEIR RELATIONS TO DISEASES OF THE EYE[1]

A Symposium

WILLIAM F. C. STEINBUGLER, M.D., HENRY W. GILLETT, D.M.D., AND HENRY S. DUNNING, D.D.S., M.D.

New York City

CONTENTS

I. The viewpoint of the ophthalmologist. *W. F. C. Steinbugler*..... 685
II. The viewpoint of the general practitioner of dentistry. *H. W. Gillett*..... 694
III. The viewpoint of the oral surgeon. *H. S. Dunning*..... 706
IV. Discussion. *Conrad Berens, J. G. Dwyer, A. H. Merritt, J. O. McCall*..... 713

I. THE VIEWPOINT OF THE OPHTHALMOLOGIST

William F. C. Steinbugler, M.D., New York City

The existence of a relationship between infected teeth and diseases of the eye has been known for some time. Several centuries ago, Hildanus reported a case of ophthalmia and loss of the eye due to an abscessed tooth. In 1795, Richter wrote regarding the connection between dental irritation and affections of the eye. In 1817 Bier described a case in which contraction of the visual field was done away with by extraction of a carious tooth. Hutchinson reported many cases of defective vision effectually relieved by the removal of pathological conditions discovered in the mouth. During the past twenty years, the subject of dental focal infection has received much attention, owing primarily to observations by Hunter of Montreal who first called attention to it in 1910, and to the experimental work of Rosenow, of the Mayo Clinic, and the clinical work of Billings. The latter had defined focal infection as a metastatic, systemic, or local condition

[1] Presented at the regular open meeting of the New York Academy of Medicine at which the New York Academy of Dentistry met jointly with the Section on Ophthalmology of the New York Academy of Medicine, at the New York Academy of Medicine, New York City, April 3, 1930.

due to infectious microörganisms or their toxins carried in the blood or the lymph stream from a focus or foci of infection. A focus of infection is a localized or circumscribed area of tissue containing pathogenic microörganisms and may be either primary or secondary.

No part of the eye seems to be immune, but the structures most frequently involved are the iris, ciliary body, and chorioid. In these locations there is a gradation from an abundant to a scanty blood supply, and hence a gradation in the supply of available oxygen. Chance describes two types of uveitis due to dental infection: (1) acute iritis with extension to the ciliary body, accompanied by intense exudation into the tissues and clouding of the sight; usually bad mouth, harboring germs; (2) low-grade chronic inflammation, no tendency to exudation; seen in the cleanly, x-ray showing apical infection. Kolmer is of the opinion that these secondary foci, in general, are due to the presence of organisms rather than to the toxins absorbed from the primary focus, while Hensen holds the opposite view, because the improvement following the removal of the toxigenic focus is so rapid. Collins believes that in inflammations of the uveal tract, the aqueous contains both the organisms and their toxins.

The frequency of dental infection can be approximated from a study of the following figures: Lang found 71 out of 176 cases, 40 per cent, due to this cause; Brown and Irons, in reporting on 100 cases of iritis in 1916, 16 per cent; in their second series of 100 cases reported in 1923, 9 per cent; Frederick, 200 cases of iritis, 37 per cent; Back, 50 cases of iridocyclitis, 32 per cent, only four in this series having been definitely negative in dental findings—all cases suspected of syphilis or tuberculosis were excluded; Byers, 80 cases of iridocyclitis, 32 per cent; Butler, 100 cases of uveitis, 12 per cent; Benedict, 60 cases of retrobulbar neuritis, 23 per cent; Elsching, 28 cases of iridocyclitis, 42 per cent; Wendt, 35 cases of iridocyclitis, 40 per cent—only three having normal teeth; Bulson, 100 cases of iritis in his private practice, 32 per cent. The French ophthalmologists, Rochon Duvigneaud, Morax, von Lint and Moreau (at a special meeting of this Section to discuss this question) considered the American concept, especially as to frequency, rather exaggerated. In only one of the above series, Back's, was the relationship of the side of the dental infection to that of the eye tabulated. In 23 cases of unilateral iridocyclitis, 8 showed

dental infection on the same side; 4 on the opposite side; 11 on both sides. In 23 cases of bilateral iridocyclitis, 9 showed unilateral infection; 14, bilateral. Of the 8 cases in which the infection occurred on the same side, in 4 the infection was in the upper jaw, in 2 in the lower jaw, and in the remaining 2 in both upper and lower jaws. These findings indicate that the infection is usually on the same side and that the upper jaw is responsible for more infection than the lower. This is probably due to the fact that caries involves the upper teeth more often than the lower.

As compared with other foci, dental infection ranks first, being followed by the tonsils, intestines, sinuses, and the genito-urinary tract in the order named. Gill, of Richmond, claims that oral infection is eight times as frequent as tonsillar infection. The dental conditions likely to form sources of infection may be grouped under three headings: Pyorrhea alveolaris, caries, and apical or periapical infection, usually in granulomata of the roots. These consist of a capsule, enclosing granulation tissue, filled with streptococci, either hemolytic or belonging to the viridans group. Of these the latter is the most frequent and most important. Periapical tissues are infected by the passage of bacteria and their toxins (1) through the apical foramina of a tooth with an infected pulp consequent as a general rule on caries; (2) from an infected periodontal membrane via the blood or lymphatic drainage of this structure; (3) in the blood or lymph circulations from more remote parts. The most dangerous forms of dental infection are those of the pulp, pulpless teeth, and apical abscesses. They are free from symptoms and hence unsuspected. Being situated in osseous tissue, no expansion can take place and their only drainage is into the circulation. They are exposed to pressure transmitted by the teeth during mastication, and as each bite has a pressure of 70 pounds to the square inch a great number of bacteria are forced into the blood stream. Impacted and ectopic teeth may, at times, give rise to inflammatory eye conditions, the infection being hematogenous, but, as a general rule, produce reflex disturbances.

The method of transmission has long been a debated question, and experimental and clinical findings have not always been in accord. Misch states that propagation by way of the lymphatics from a tooth to the eye would always have to follow a round-about course through

the pre-auricular lymph glands. Birch-Hirschfeld found a system of spaces in the orbital tissue, which is to be regarded as a lymph space system. But a communication of the lymph vessels of the orbit with those of the antrum of Highmore, or the other accessory sinuses has, so far, not been demonstrated anatomically. Through the study of numerous microscopical preparations from a case of orbital phlegmon, he is convinced that these spaces serve as routes for the invading leucocytes and lymphocytes. Yet he assumes a thrombophlebitis, phlebitis, or periphlebitis, in practically all cases where infections are transmitted from the orbit to the meninges; and he opposes the belief of other authors that the processes may spread along the nerve sheaths. Hensen believes that the transmission is either through a thrombophlebitis or the antrum of Highmore, the former when the canines and bicuspids are involved, the latter from the molars; and that iritis is probably caused through lymph-channel transmission. Maiden gives four routes of transmission to the eye: by continuity; by way of blood vessels; by way of lymphatics; by way of lymph sheaths of the nerves. Back claims that transmission can take place along the nerves, as evidenced by the spread of the virus of herpes from the second branch of the trigeminus to the first. Benedict recognizes the following methods of transfer to the eye: (1) directly through bone— the tooth socket becomes the seat of an infection that destroys the bone of the upper jaw, and through that directly affecting the orbital walls, finally producing a cellulitis or retrobulbar neuritis; (2) direct extension of the process along the periosteum to the malar bone, invading the orbit and giving rise to similar processes; (3) transfer of organisms from the focus at the tooth to the eye by the blood stream.

Of interest in this connection is the experimental work of Rosenow in establishing his theory of elective localization. Latent foci were produced by devitalizing and infecting the teeth in dogs, closely simulating the conditions often observed in persons. Nephritis, nephrolithiasis, and ulcer of the stomach, have been produced with cultures isolated from patients having these respective diseases. In each instance, the causal relationship between the organism introduced into the teeth and the metastatic lesion was established by the demonstration of the organism in the lesion, and by the elective localizing power of the strains isolated at the end of the experimental period. Of 66

guinea pigs injected intravenously with cultures obtained from dental granulomata of patients having eye conditions, 66.7 per cent had metastatic eye infections. Of 189 guinea pigs injected in the same manner, using cultures obtained from teeth of persons having no ophthalmic conditions, 14 per cent showed metastatic eye lesions. Back studied the material in Elschnig's Clinic at Prague over a period of three years in order to determine whether he could corroborate Rosenow's findings from a clinical standpoint. Only chronic cases with purulent processes (granulomata) on the apices of the teeth were included. Fifty-six were examined and cultures made. The usual precautions were taken regarding the extraction and the granulomata were clipped off with sterile scissors, opened up, and their contents cultured. The great majority showed streptococci, but a 24-hour culture injected hypodermically into mice produced no results. Fourteen rabbits were injected with cultures taken from the teeth of patients suffering from iridocyclitis, but in none of them did any eye trouble develop. Moody, in his experiments on 78 animals injected with streptococcic cultures from teeth of patients suffering from iridocyclitis, was able to obtain only 1.7 per cent of positive results. These experiments indicate that direct metastasis through the blood does not take place as frequently as Rosenow and his followers have reported. Noyes and Ladd, in a study of the lymphatics of the dental region, injected the lower canine teeth of dogs with ferric ammonium citrate. The teeth were trephined and the fluid injected through the dental tubules into the pulp chamber. The fluid came out through the inferior dental canal, the reaction with potassium ferrocyanide being employed to demonstrate its presence. The first gland of the inferior maxillary group on the same side dissected out, also showed the presence of the injected fluid. The corresponding gland on the opposite side gave a weaker reaction at the end of forty-eight hours. This would prove that most of the dental infection is carried by the lymph channels.

From a clinical standpoint, a sufficient number of cases have been reported to lend support to the belief that the method of transmission is variable. Gutman reports the cure of a fistula in the lower lid as the result of removal of the upper central and lateral incisors on the same side. The cure is explained on the probability that bony

channels extend from the alveolar processes of the upper jaw to the lower margin of the orbit. Hirsch reports a case of complete blindness following extraction of the second upper left bicuspid. There was a purulent alveolar periostitis of the anterior surface of the superior maxilla, which extended over the lower margin into the orbit, giving rise to inflammation of the connective tissue, optic neuritis, and finally atrophy.

Of the organisms responsible for dental infection, streptococci are by far the most frequent in cultures of tooth roots. The nonhemolytic variety is present in the great majority of cases, the hemolytic type being encountered rarely. Pneumococci, staphylococci, and Micrococcus catarrhalis occasionally act as causative factors. In Rosenow's experimental work the microörganism that manifested the greatest elective localizing power, and with which the common forms of disease of the eye have been reproduced, was a streptococcus that usually produced greenish or slightly hemolytic coloring on blood agar and which required a reduced oxygen tension for its isolation. Haden undertook an extensive bacteriological study of chronic periapical dental infection. His conclusions, based on cultures from the apex and periapical tissues of 1500 teeth, have been reported. The teeth were extracted under sterile conditions. The granulomata were cut off with sterile scissors, and dropped directly into a tube containing about 1 cc. of sterile 0.85 per cent sodium chloride solution and a small amount of sharp sand. Glucose brain agar and broth were used as culture medium. Of 1091 broth cultures, 963 showed pure growths of streptococci; 37, streptococci mixed with gram-positive bacilli. In over 92 per cent of the cultures, streptococci were found either alone, or mixed with other bacteria. Of 346 of the cultures transferred to blood-agar plates, only 3 contained hemolytic streptococci, which were found twice in pure cultures, and once associated with Staphylococcus aureus. The most common types of streptococci, as determined by the carbohydrate fermentation test, are the fecalis, mitis, and salivarius.

Okumura and Nakai, in studying dental decay with particular reference to the specific parts played by certain bacteria, found that acid-producing organisms decalcify the hard dental tissues and prepare the teeth for subsequent invasion by various germs; and that the

latter, in turn, are responsible for the caries. These are most commonly Streptococcus brevis, Staphylococcus albus, and a gram-positive bacillus named Bacillus necrodentalis. Bulleid finds the most common cause of dental infection to be the non-hemolytic or viridans type of streptococcus, followed in the order of frequency by Staphylococcus albus most frequently; aureus, rarely; and by gram-positive bacilli. Back, in studying the material from Elschnig's Clinic at Prague, already referred to, examined 56 cases of granuloma bacteriologically. The technic of preparation and extraction differed little from that of Haden. The contents of the sac were cultured; blood serum bouillon and the anaerobic method of Kitt and Tarozzi were employed. Forty-seven cases showed pure cultures of streptococcus; 9, mixtures of streptococcus and staphylococcus. In 12 cases, hemolyticus was present four times—in the remaining eight, one of the viridans group was found. Regarding the pathogenicity of cultures from dental foci, rabbits were injected with the original broth cultures from the root tip. All types of lesions that can be produced by the intravenous dissemination of bacteria were seen. The most common finding was a joint lesion followed by lesions of the kidney, endocardium, myocardium, brain, eye, stomach, and duodenum.

In the study of the relationship between dental infection and diseases of the eye, the x-ray has been a valuable aid. Bulleid concluded that of root-filled teeth, those most likely to show rarefied areas are the ones with good root-filling to or nearly to the apex. Teeth most likely to show no apical shadows in skiagrams are those with no root fillings or only partially filled roots. Of extracted teeth, the only ones that showed granulomata were those with no root fillings or only partially filled root canals. Haden made a study of cultures obtained from the periapical tissue of 1307 vital and pulpless teeth, and compared them with x-ray findings. The technic employed provided (1) proper controls to determine the chances of error; (2) a medium exceedingly favorable for the growth of the organisms usually encountered; (3) a means of measuring the extent of infection by determining the number of bacteria in the tissue culture. Of 392 vital teeth, 9 per cent showed from 1 to 10 colonies in a dip-agar tube; 5 per cent, 10 or more colonies; and only 1 per cent over 100 colonies. Of 490 pulpless teeth,

negative in the radiograph, 10 per cent showed from 1 to 10 colonies; 44 per cent, 10 or more; 24 per cent, 100 colonies. Of 425 pulpless teeth positive in the radiograph, 10 per cent showed from 1 to 10 colonies, 60 per cent, 10 or more colonies; 44 per cent, over 100 colonies. He concluded that the incidence of infection is almost as high in the radiographically negative group as in the positive. There is a very sharp limitation to the translation of radiographic evidence of infection into terms of bacteria. The absence of radiographic evidence of infection at the apex of a pulpless tooth never excludes the presence of active infection. In many cases the radiographically negative tooth is a far greater source of systemic infection than the positive, since in the former there may be little resistance to the infection.

Regarding the use of vaccines and their value from the clinical standpoint, there is wide diversity of opinion. Lanier, in a series of infective eye diseases treated during 1920–1921, obtained more favorable results from the injection of stock vaccines or serums, both the specific and para-specific products having been employed. Weaver thinks that streptococcus vaccines should always be autogenous; and that, in general, autogenous bacterins are to be preferred to the stock preparations. The best results have been obtained when the pus has been recultured, and fresh bacterin prepared every two to four weeks. In cases of iridocyclitis, cultures may be made from the aqueous. The vaccines prepared from these have given excellent results. Rosenow has obtained favorable results with vaccines prepared from the cultured strain, but warns against expecting too much from the use of specific vaccines or serums, since the diseases that commonly result from focal infection are usually due to streptococci, immunity to which is of short duration. In a small series of cases in which vaccines were employed, he reports 26 per cent improved, 46 per cent unchanged, and 28 per cent worse, compared with 21 per cent improved, 32 per cent unchanged, and 47 per cent worse, where no vaccines were used. Berens believed that patients who had been treated with vaccines, made from organisms to which they were particularly hypersensitive, were benefited more than those treated with autogenous mixed vaccines. But when nineteen patients treated with stock,

mixed, catarrhal vaccines for control were compared with nineteen treated with selected autogenous vaccines, the results were identical. He concluded that the non-specific factor is extremely important. Benedict and De Schweinitz both report gratifying results from the use of autogenous vaccine.

In a study of the blood in cases of dental infection, Toren found a cell which he called a specific type of lymphocyte, demonstrable by a special type of stainability. It is described as a large lymphocyte, the outline of which is usually irregular; the cytoplasm is not the robin-egg blue of the normal large lymphocyte, but instead has a peculiar greyish grainy appearance. The nucleus is always irregular in contour, and stains with a moderate degree of intensity. It is frequently cuboid or ovoid with one flattened side. The irregularity in the shape of the nucleus does not correspond with the irregularity in the shape of the cytoplasm—is not due to pressure from adjacent cells. These lymphocytes have never been found in the blood of a patient who did not have oral infection, nor has a case of oral infection been seen in which these lymphocytes were absent. There is much doubt about this cell being a lymphocyte, since it has the characteristics of the monocyte described by Sabin. Walter found these cells in 98 per cent of cases examined. Up to 1.5 per cent these cells are normal, above that figure pathological. Other findings in these cases were low normal or low white blood-cell count—relative leucopenia; low red blood-cell count; relatively low "poly-count"; relatively high lymphocyte-count; low thyroid activity; low pulse rate; subnormal temperature, and low blood calcium.

No definite clinical ophthalmic picture results from dental infection. Focal infections, of which the dental form the largest group, must be considered as etiological factors ranking in importance next to syphilis and tuberculosis. When a suspected focus has been removed, and a rapid and marked lessening of the inflammation does not set in within forty-eight hours, the true cause has not been reached and a further search should be made. Finally, while, as a general rule, conservatism should be practised regarding the extraction of teeth, in doubtful cases the eye should be given first consideration, and elimination of oral sepsis given precedence over preservation of the teeth.

II. THE VIEWPOINT OF THE GENERAL PRACTITIONER OF DENTISTRY

Henry W. Gillett, D.M.D., F.A.C.D., New York City

The present trend of dental thought and research is strongly towards prevention of oral disease, and for that part of the juvenile public which cooperates much of the familiar corrective labor of the dentist is becoming less necessary. Further discoveries in prevention seem imminent, and increased interest of the medical profession, in the one organ concerned in nutrition whose functional activity is largely controllable by the individual, will be a potent influence in furthering public health.

There is plenty of evidence of a direct etiologic relation of mouth foci to inflammatory eye disturbance, in the case histories of both sections of this conference. In this effort to establish a better contact between these two divisions of health service, we are confronted with the fact that those in each division are usually poorly informed concerning the anatomy, pathology, and the function of the organs in the care of the other. This is a specially unfortunate status as regards the mouth, because (1) it is the portal by which food normally reaches the organism, and nutrition is fundamental to all health. (2) The normal or abnormal development of teeth and jaws influences in large measure the size of the external air passages, and determines whether the tongue shall occupy its normal position, or be forced back into the pharynx with resultant interference with the respiratory function, which is another health fundamental. (3) It is the portal of entrance for a large percentage of the infective factors reaching the organism from the outside, and in it are many separate potential locations for the development of foci of infection together with many conditions favoring the proliferation of bacteria. (4) The relation of the mouth to the rest of the digestive tract insures distribution through the tract of infectious material from the former. (5) The mouth, in its relation to the respiratory tract, frequently facilitates the transfer of infection to the bronchi and lungs. (6) The relations of lymphatic drainage and blood supply of the teeth and gingivae favor easy distribution of organisms and toxins from regional foci to the lymphatic system and the blood stream. (7) Effective functional use of the teeth, in the initial step of digestion, compels forcible introduction of filth and

organisms from foci about the teeth into the adjacent vascular tissues, and into their lymph capillaries.

The macroscopic anatomy, the functions, and the special pathology of the dental apparatus are largely a closed book to other specialists because medical curricula rarely include more than perfunctory mention of them. With the approval of your President, I present a slide with a brief study of mouth and tooth anatomy. Your attention is called to the following seven features: enamel, dentin, cementum, pericementum, alveolar bone, pulp, gingival crevice, and relations of margins of the gingivae. Four of these—pulp, pericementum, gingivae, and cementum—are intimately related to the mouth foci of infection and a fifth—dentin—is often contributory.

The tooth pulp is often erroneously called the "nerve," even by dentists. It would be equally correct to call the whole eye a nerve. The pulp contains connective tissue, specialized cells, nerves, and blood and lymph capillaries. There is no collateral circulation, but the pulp in each root of multi-rooted teeth, and a part of the bulbous portion closely associated with it, appear to have a vascular and probably a lymphatic system independent of that in the others. In its first stage, it was the formative organ for the dentin, enclosing itself except for foramina at or near the root apex, and it remains as its nutrient organ. When it is dead, or moribund beyond capacity to function, the tooth should be called a "pulpless" tooth, never a "dead" tooth, unless the pericementum is also dead. Its nerves register acute pain when irritated, but they are not concerned in the modified tactile sense of teeth. When a pulp dies, it usually decomposes like any bit of necrosed vascular tissue. The spreading of toxins and bacteria involved in this decomposition causes many of the apical foci. Infected *vital* pulps, whose circulation and nerve function continue active, are potential foci for metastatic disease, and a greater menace than apical foci, because they are usually more difficult to diagnose accurately. They frequently occur from penetration of streptococci to closed pulp chambers through normal-appearing dentin without pain association or discoverable physical changes. According to Thoma (4), "infected pulps may remain vital for a long time, and meanwhile be a focus from which continuous absorption of bacteria and their toxins may take place." His tabulation of cases supports his conclusions. Hope-

well-Smith (5) says: "The tubes of dentin are admirably adapted for rapid and complete infection; those of the coronal region especially. The tubular nature of dentin, and the character of the contents of the millions of channels pervading it, present very favorable conditions for pulp infection. This is a matter of common knowledge, but the well authenticated fact, proved by sections and photomicrographs, that infection of the pulp commonly occurs *before as well as during actual exposure in deep seated cavities, is not so well known.*"

Cementum is a thin layer of bonelike material covering the root. It is incapable of self-repair and, when infected, is difficult to sterilize. The pericementum is an extension of the periosteum, is richly supplied with nerves, and blood and lymph capillaries, and is the developmental, nutrient, and reparative organ for the cementum. Its diagonally placed, interlocking bundles of ligamentous fibres attach the tooth to the lamina dura of the tooth sockets, and provide an elastic support to absorb the shocks of functional use of the teeth. Through its nerves, we are conscious of the contact of teeth with each other and with other substances. Its blood and lymph capillaries connect freely with those of the pulp, the gingival crevice, and the surrounding alveolar bone. Under physiological conditions, it repairs damaged cementum in periodontoclasia pockets and establishes physiological union with it, and it may seal-in a dead pulp by closing the apical foramen with new cementum. Coolidge (2) has recently reported a case of complete coverage of the dentin of a resected root-end with new cementum. When pulp infection or necrosis causes infiltration of periapical tissues, thickening of the cementum is usually the first physical change discoverable radiographically. A periapical focus may exist without causing enough physical change in the pericementum to register in an x-ray film. Periodontal abscess may occur without pulp infection, probably from infection of lymph capillaries from the gingival crevice. They are potential foci of infection.

The free margin of the gingivae surrounds the teeth like a collar, forming a crevice 1/16 inch deep, or less. The epithelial covering of the outer gingival surface is thick and tough, that of the inner surface very thin; lymph capillaries apparently open into it, and also connect with the pericemental vessels. Other potential foci are abscesses in the gingivae. They are easily located, and usually yield promptly to indicated treatment.

Infective foci often occur in the pulp as a result of caries. The resulting pulp abscess or other lesion may develop on the surface, or within its substance. Ordinarily, this leads to death of the pulp, but Thoma (3) has demonstrated that re-solution can occur in pulp abscesses. The pulp then continues to function for a considerable period in teeth whose pulp chambers have not been penetrated by caries. Exact information as to the complete pathological picture from the first slight infection, to establishment of a definite pulp focus and then pulp death, is difficult to obtain, because the pulp tissues are so effectively covered from observation as to completely mask the conditions until they reach an advanced stage. Clinical observation and interpretation indicate that the progress from mere abnormality and stimulation of protective activity that deposits adventitious dentin, because of beginning invasion by toxins and bacteria, on through hyperemia, congestion, inflammation, and stasis, to a moribund condition, may be spread over many weeks or even months. The pathogenic organisms of these pulp abscesses and their products are in close relation to the engorged capillary and lymph vessels. Cases reported by Thoma (4) and Rickert (5) indicate that the infected vital pulp is a rather frequent focus in eye lesions. Only one of the eight cases reported in Thoma's article was related to the eye, but the list is instructive. It includes one case each of symptoms of general toxemia and joint lameness; chronic exhaustion and temperature rise; multiple arthritis; swollen, painful extremities; retrobulbar neuritis; high blood-pressure; pain and swelling in right wrist and neuralgic pain. In all these cases, the primary focus was within the pulp chamber, where its presence would be recognized only by an expert interpreter of exceptionally good radiographs of individual teeth, coupled with intelligent correlation of the strictly dental physical conditions and history. All the cases either recovered promptly, or showed marked improvement, on removal of the mouth foci.

It should be borne in mind that most dentists can supply case histories in which removal of suspected teeth has brought no improvement in the conditions for which they were held to be responsible, and where their loss caused a major disability for the patient. One of the saddest experiences of my professional life was my acquiescence in the removal of several teeth for a woman of most lovable character

and great prominence, without the slightest discoverable benefit. No tooth was removed without proof of the presence of pathogenic organisms in its periapical tissues. I now believe that the internist was groping in the dark in a last stand against conditions in which removal of foci was futile, and that her last three years were made desperately unhappy because of inadequate diagnosis and honest but mistaken professional zeal.

Reflex pain on any branch of the fifth nerve is not uncommon when the effort of an inflamed pulp to swell within its chamber, or when adventitious dentin nodules, cause irritation of pulpal nerves.

A common history of progress of pulp infections is (*a*) slow infiltration through the pulp tissue to the pericementum of the apical area while the pulp is still alive, usually associated with streptococcus infection; or (*b*) death of the whole pulp before the disease extends beyond the root apex, usually associated with staphylococcus or mixed infections which set up inflammatory reactions that strangulate the circulation at the foramina. In either case, an additional focus is often established in the periapical tissues. When staphylococci are among the invading organisms, active inflammation and suppuration usually occur, pus burrows through the tissue, the face swells, and the pus finally evacuates with or without interference. The fistula from such an abscess ordinarily remains open, and steadily discharges small quantities of pus into the mouth; or the mucous membrane may, from time to time, close over the opening, in which case the chronic "gum boil" and periodical discharge will occur. Very often the organism involved is Streptococcus viridans, and then the local irritation is slight. After pulp death as a result of infection, infiltration of the tissues commonly results in (*a*) the development of a small cavity in the alveolar bone around the root, within which the involved tissues are often encapsulated. A condensing osteitis may result in the development of a narrow zone of dense bone around them, which shows up as a white line in the radiograph; or, (*b*) the organisms may spread into the alveolar bone and set up changes registering in the x-ray as a dark area of irregular form. These conditions may persist for years without enough local disturbance, other than occasional slight tenderness, to attract attention. The secondary local condition, which carries the lesion definitely beyond the range of conservative treat-

ment, is the stripping of the peridental membrane from the cementum and the infection of the latter. Infected cementum in this region cannot be sterilized, and it cannot be exfoliated until the whole tooth goes with it. In single-rooted teeth, resection of the root-end may properly be considered, but in multirooted teeth extraction is the correct operative procedure when a root tip has become denuded. Poor extraction technique may leave the infected tips of roots, or other infected tissue, in the wound, where they may continue to act as foci.

Most pulp infections are due to caries, and much of the caries causing them has its beginning in pits or fissures, which are developmental defects. Here is the real battleground for prevention of pulpal and apical foci. The child for whom each tooth receives protective treatment within six weeks of its eruption, who is effectively taught adequate mouth hygiene, should have little, if any, trouble through life from pulpal foci or periodontoclasia. Just peeping over the horizon are indications that prevention of developmental fissures and, when they are present, prevention of caries in them by nutritional practice, may soon be possible. If this happens, responsibility for the occurrence of pulpal and apical foci will rest upon the shoulders of the parent.

Toxic and bacterial penetration of the dental tubuli may be rapid. In the deciduous teeth of children, the distance between the floor of a carious cavity and the pulp chamber is short. In all cases of eye disease that may be of focal origin, and especially for children, there should be careful search for infected vital pulps. This involves search for possible hidden caries as well as close scrutiny of all fillings. When extraction of suspected pulpless teeth results in no benefit for a secondary infection probably due to mouth foci, it should prompt intensive search for infected vital teeth.

Where organisms in the pulp chamber set up inflammatory action, or cause gas formation, they are forced from the infected pulp chamber into the periapical tissues. After spontaneous pulp death, there is usually seepage through the foramina into the periapical tissues. In the absence of corrective treatment, the decomposing remains of the pulp and of the fibrillae in the dentin are a continuing reservoir of chronic infection. The dental view has been that complete removal of the remains of the pulp, sterilization of the pulp chamber, sterilization

of the dental tubuli so far as possible, and complete closure of the chamber with inert material, make it safe to retain such teeth in the mouth. To do this effectively taxes the utmost skill of the dentist. Much incomplete and slipshod work has been done in this field, and is largely responsible for the heated arguments as to whether any pulpless tooth may be safely retained. Conservative dental opinion does not yet sanction extraction when pulp infection or pulp death are discovered, if adequate treatment is carried out before tissues outside the tooth become pathological beyond their capacity to recover. If uveitis and iritis promptly recover after removal of a tooth-pulp focus etiologic to them, it is rational to expect well advanced pericementitis to do the same.

The reluctance of the dentist to extract suspected pulpless teeth is frequently misunderstood and criticized. When the case history includes grave secondary disease probably or possibly due to infection, and no other focus can be located, then any or all teeth possibly involved must be sacrificed unless their condition is such that probable foci can be quickly removed by less extreme measures. Many millions of pulpless teeth are now functioning effectively in mouths of persons in perfect health, as indicated by competent physical examination. This tends to support the conservative view that radical action, which would set up a permanent and often grave disability in an important organ, because of apprehension about disease that is believed to occur in a small proportion of those carrying the potential possibility, is ill advised for a person in sound health. The extraction of a single tooth from an already mutilated denture may change its status from one capable of being maintained in efficient function to one so inefficient that entire artificial dentures become essential. The extraction of a single tooth from a sound denture, and neglect to promptly fill its place with a substitute, may result in the gradual establishment of serious functional inefficiency of the whole masticating organ.

The mouths of clinic and hospital patients are frequently very foul, and the gingival crevice and pus pockets that usually occur as extensions of this condition are always seething with bacteria. Their close relation to the lymphatic drainage leads me to mention this condition as worthy of consideration when seeking the location of primary infec-

tion. In that connection, I want to record here the opinion that any surgeon who permits the administration of a general inhalation anesthetic in a person having a filthy mouth accepts a grave risk of serious respiratory complications sometimes culminating in lung abscesses, and perhaps of being compelled to write in his case history: "Operation successful; patient dead."

Periodontoclasis has not yet received sufficient emphasis as a probable infective focus, but some of those to follow me will deal with it better than I can. Noyes and Dewey (6) quote Ollendorf as having reported enlarged glands in all but three of 2246 patients with periodontitis, evidently using the term as equivalent to periodontoclasia in our present terminology. Haden (7), in his excellent book regarding association of chronic oral infection and chronic disease, reports a series of forty-seven ophthalmic cases associated with mouth foci, in which he found three-quarters of the foci were periapical and one-quarter gingival; another from Irons (8), in which 36 per cent of forty-seven patients with iritis had periapical infections. Hartzell (9), a physician and dental specialist in periodontoclasia, states that a case of advanced periodontoclasia may present the equivalent of approximately seven square inches of ulcerating surface from which the lymphatics, veins, and blood stream can be and are invaded. He regards periodontoclasia, which under present hospital routine frequently exists without the knowledge of either physician or dentist, as a frequent cause of leukopenia and secondary anemia. When periodontoclasia reaches an advanced stage, there is more than opportunity for the spread of infection from its pus pockets—there is a driving force due to ordinary functional use of the teeth that recurrently compels migration into the surrounding vascular tissues.

In a recent paper under the title, "Focal infection from the ophthalmologist's standpoint," Ring, (11) reports a case of interest. After saying that periapical infection is probably the most frequent cause of secondary manifestation of local infection, Ring states his position as follows: "No operative case in ophthalmology, which involves the opening of the eyeball, should ever be undertaken without a complete dental study. An exception to this dictum that I would permit would be such a case as one of acute inflammatory glaucoma, or one associating ocular with serious sinus disease." The high points of the

case referred to are as follows: Age 65, acute uveitis, vision hazy 20/200. White-cell count of 13,000, and 6 infected teeth. He concludes: "The case strikingly illustrated also the exacerbation of the symptomatology following the sudden pouring into the blood stream of an additional group of viridans streptococci upon the extraction of the first two of six teeth. The same experience was repeated a week later when the third and fourth teeth were extracted. By this period the autogenous vaccine was being administered and when still a week later the last two offenders were sacrificed, the resistance had been raised, the inflammatory symptoms rapidly subsided, and the vision returned to normal. This, briefly, was the dominant symptomatology in an eye headed for disaster, unless the blood stream leading had been followed to its toxic foci, upon the elimination of which a perfect result was produced." Ring reports three other cases of uveitis, all with a similar blood picture, the presence of infected teeth, and a happy result.

With full realization of the gravity of the secondary disturbance that may result from the retention of undetected foci about pulpless teeth, I am satisfied that the public welfare will not be served by indiscriminate removal of *all* pulpless teeth, as advised by some practitioners. But in the present state of our knowledge, all of them should be kept under observation. Numerous observers have recorded the presence of pathogenic bacteria in pulps and in apical areas in which no indication of pathology or lowered function could be detected. In the absence of definite evidence of the presence of a focus, I would counsel a conservative attitude by all practitioners of medicine in urging the removal of teeth regarded as important by a well qualified dental consultant familiar with root-therapy practice. Diagnosticians and oral surgeons constantly see cases in which tooth infection has caused disturbance, but lack opportunity to evaluate the status of the much larger number of pulpless teeth that cause no trouble and are valuable functioning units in their respective dentures. This fact leads to a considerable divergence of opinion between them and general practitioners of dentistry. There is little doubt that the general practitioner often overestimates the importance of certain teeth, but in my opinion the tendency of the surgeon and diagnostician, to class all pulpless teeth as dangerous, is influenced by their restricted opportunity for observation of the whole field.

Hanke's (10) demonstration that the local resistance of gingival tissues already attacked by pathogenic organisms can be built up quickly by simple nutrition therapy, to a status enabling it to repel the attack without local treatment, should be tested in other fields; it might induce considerable revision of the present evaluation of simple foci. Osgood (12), speaking from the orthopedic viewpoint, counsels "conservatism in surgical and dental removal of foci not proven to be affecting the general health of the patient unless sure they are not simply themselves expressions of a lowered vitality for which there are other causes." Hanke appears to have shown that in the gingivae, the first stage of an important group of mouth foci is a local expression of lowered vitality caused by nutritional imbalance of a type usually easy to correct.

Up to 1911, the presence of lymphatics in the dental pulp was denied. Noyes (13) has recently shown that lymphatics draining the pulps and pericemental tissues of teeth in the maxilla join the drainage leading through the infraorbital canal, while in the mandible they pass to the inferior dental canal, and that from both areas they reach the first submaxillary lymph node. His latest demonstration proves that fluids can be passed through the tubuli of sound teeth into the pulps. Applebaum (14), working under Bödecker, and Fish (15), have reached the same conclusion. Bödecker, Applebaum, and Fish consider the fluid content of the tubuli to be lymph, while Noyes leaves the question open. The fact that liquefaction of the organic content of dental tubules is all that is needed to permit direct migration of organisms through them to the lymph capillaries, puts the carious tooth-cavity on the list of potential infective foci, lends emphasis to the importance of the search for an effective means of preventing dental caries, and places upon the dentist the responsibility of protecting the exposed ends of tubuli in vital dentin from possible infection during operative procedure.

Hanke, Price, Davis, Drain, Boyd, Bunting and others, each working by a different technique, all seem to be approaching a tentative conclusion that nutrition, optimal in quality, and freedom of the organism from overload of surplus food and waste products, are the really important factors in the prevention of dental disease. Is it just possible that the connection between the mouth and the field of every other

specialty is the extreme popularity of that age-old entertainment, the tickling of the palate with an attractive but unbalanced diet, bringing in its train susceptibility to the attacks of the myriads of microörganisms that are always with us? If research findings indicating that right nutrition can render gingival mucous membrane immune to bacterial attack are correct, as now appears probable, are not other membranes logical fields for similar research? Tooth pulps will surely be subjected to such a study. I venture no hypothesis about eyes, but I think all will admit the soundness of the principle of building up resistance in any organ or tissue, where possible to do so. A healthy and effectively functioning masticating organ is an essential in establishing optimal nutrition.

In this connection, it is to be noted that several investigators have stated that Streptococcus viridans can be cultured from vital and apparently healthy pulps of all teeth that are carious, and of all those that have periodontoclasia in moderate degree. It has been amply proven that organisms and toxins from all mouth foci, including the carious cavity and the gingival pus-pocket, have a direct drainage path through the lymph channels to the blood stream. The ophthalmologist will evaluate these facts and apply them to his own field of work. For the dentist, they mean increasing vigilance in preventing the establishment of foci, and intensified interest in intelligent coöperation with the ophthalmologist whenever a search for primary foci responsible for eye disease is indicated. Rickert (5) quotes one New York dentist as having written to him, on the publication of his first article concerning degenerating vital pulps, as follows: "You may be right about the seriousness of the infected degenerating pulp, but soft pedal on the subject, for if the medical profession ever gets the idea that vital teeth may, at times, be infected and a menace to general health, Heaven help us." We venture to hope that this was half humorous, and only the result of exasperation after an unhappy experience in consultation. Whether it was or not, the dental group in this conference is eager to have the medical profession know all the facts, because a full realization of them must lead the medical profession to give active support in the campaign to teach the public that at least half the potential dental ills of the child who starts right, and plays the game loyally, are preventable throughout life; also active

coöperation in, and support of, the present search for more effective means to limit the incidence of caries and periodontoclasia.

My concluding word to this group is this: In the effort to serve the public, with due regard to the best health service principles, the dentist badly needs coöperation from the ophthalmologist in order to insure better understanding of conditions that are of mutual interest. The public welfare will be served, if the ophthalmologist weighs with deliberation and care the views of the conservative dentist concerning conditions in the mouth and his estimate of the values of radical surgery in that field. The following is a summary of my conclusions based on reported case histories.

1. Any variety of mouth focus may be a cause of eye disease.

2. Infected vital tooth pulps, apical areas, periodontoclasia areas, and impacted teeth, are the four most common mouth foci to be considered.

3. A negative x-ray report of a pulpless tooth is inconclusive, if there is reason to suspect a tooth focus.

4. If a tooth focus is suspected, a negative x-ray report plus a negative physical mouth examination, call for keen search for infected vital pulps.

5. If streptococcal infection is suspected, absence of tooth pain does not eliminate tooth foci, but should spur a keener search for infected vital pulps.

6. The lymph and blood circulation of the tooth pulp and periodontal tissues suggests that this is the path by which infection from tooth foci reaches the eye. Case histories in which mandibular teeth were the source strongly tend to confirm this view.

References to literature

(1) Noyes, F. B. 1930 A textbook of dental histology and embryology, 4th ed., p. 157; Lea and Febiger, Philadelphia.

(2) Coolidge, E. D. 1930 Root resection as a cure for chronic periapical infection. *Journal of the American Dental Association*, xvii, pp. 239–249.

(3) Thoma, K. H. 1929 A comparison of clinical, roentgen, and microscopical findings in fifteen cases of infected vital pulps. *Journal of Dental Research*, ix, pp. 447–487.

(4) ——— 1928 An important focus of systemic disease. *Ibid.*, viii, pp. 529–550.

(5) Rickert, U. G. 1927 The clinical significance of the infected degenerating pulps. *Journal of the American Dental Association*, xiv, pp. 1895–1900.

(6) DEWEY, K. W. 1918 Lymphatics of the dental region. *Journal of the American Medical Association*, lxxi, pp. 1179–1184.
(7) HADEN, R. L. 1928 Dental infection and systemic diseases, p. 33; Lea and Febiger, Philadelphia.
(8) IRONS, E. E. 1916 Dental infections and systemic disease. *Journal of the American Medical Association*, lxvii, p. 851.
(9) HARTZELL, T. B. 1929 Interesting cases of advanced periodontoclasia before and after treatment. *Journal of the American Dental Association*, xvi, pp. 1307–1308.
(10) HANKE, M. T. 1929 The relation of diet to caries, and other dental disorders. *Journal of the American Dental Association*, xvi, p. 2263; also a recent unpublished paper.
(11) RING, G. O. 1930 Focal infection from the ophthalmologist's standpoint. *Dental Cosmos*, lxxii, pp. 280–283.
(12) OSGOOD, R. B. 1926 Orthopedic aspects of chronic arthritis. *Journal of Bone and Joint Surgery*, viii, pp. 1–41.
(13) NOYES, F. B. AND LADD, R. L. 1929 The lymphatics of the dental region. *Dental Cosmos*, lxxi, pp. 1041–1047.
(14) APPLEBAUM, EDMUND 1929 Lymph channels in dentine and enamel stained by amalgam. *Journal of Dental Research*, ix, pp. 487–502.
(15) FISH, E. W. 1927 The circulation of lymph in dentin and enamel. *Journal of the American Dental Association*, xiv, pp. 804–817.

III. THE VIEWPOINT OF THE ORAL SURGEON

Henry Sage Dunning, D.D.S., M.D., B.S., F.A.C.S., New York City

My remarks must from necessity be chiefly clinical in character, as it seems almost impossible these days to be a clinician and a research worker at the same time. For many years, I have been calling the oral cavity the dirtiest cavity in the body, and have claimed that the maxillary bones and the mandible become infected more often than any bones in the body. This statement caused little or no comment until last year, when it was challenged by my friend, Dr. Anthony Bassler, who made me feel very badly when, at a dental meeting in Connecticut, in his scholarly way, he called attention to the fact that the intestines are much more unclean, and that they contain about 260 different kinds of "*bugs*." Of course, I felt disappointed because I knew that we could find in the mouth only about 112 different types of bacteria. If Dr. Bassler had stopped there, I should have hobbled off then and quit getting excited about the dirty mouth. But he went on to say that nearly all pulpless teeth became non-vital because intestinal bacteria had a predilection for pulps of teeth whose vitality was

low, and that they found their way thereto through the blood stream, causing the death of the teeth by their action; also that more teeth become non-vital in this manner than from caries. I feel very strongly that teeth become infected in *three* ways, namely, the *intra-dental route*, that is, decay within the tooth involving the pulp and passing through the pulp canal to and through the apex of the tooth. This causes the true apical infection. Next, the *peridental route* under the gingivae, stripping up the peridental attachment into the alveolus, and bacteria gaining access to the bone and lodging there. *Lastly, through the blood stream.* I believe that *the two dental routes* are much more frequent than the third or hematogenous path, and that this can be proven by thousands of radiographs in the possession of the dental men in this audience.

If we assume that these dental routes exist, can we not easily see why the teeth and the mouth are so important as a port of entry of bacteria into the system? If we have thirty-two teeth in a given mouth, we have thirty-two chances of bacteria entering their pulps and gaining access into the spongy vascular bones of the jaws—the *intra-dental route.* If we have thirty-two teeth in the mouth, we also have thirty-two and many more than thirty-two chances of the delicate gingival flap becoming traumatized by calcareous deposits and what not, giving the bacteria in the mouth free access into the alveolus by stripping up the peridental membrane—*the periodental route.* Lowered resistance of the *local tissue*, of course, plays an important part in the above picture as does lowered resistance of the patient in general. When bacteria enter the spongy cancellous bones of the jaws, they find there proper nourishment and environment, and remain for years—in many cases, I believe, for the life of the patient. *Question:* How can we sterilize the bones of the jaws, and how can we prevent this infection of the bones from causing some systemic disturbance? This is the real crux of the dental situation as I see it. Pus under pressure, locked up in spongy vascular bone, giving no trouble for years, but continuously and *relentlessly* breaking down the patient's resistance until his body chemistry has undergone certain changes that, as yet, we do not know all about. Secondary changes then take place in the blood and perhaps in all other tissues including the eye, the organ of special interest to us tonight.

In comparing the two routes by which the bone becomes infected, I would say that the intra-dental route through the dental canal produces the most serious of the three classes of infection. A very high percentage of all apical infections, when the teeth are removed without contamination, show pure cultures of Streptococcus viridans, hemolytic or non-hemolytic in character. Pure strains of Streptococcus viridans are obtained so often from pulpless teeth that I think nowadays we do not consider it important to culture teeth in the routine way of yesterday, knowing that most pulpless teeth will give us a culture of this streptococcus. Cultures taken from periodontal lesions generally show quite a variety of bacteria including staphylococcus, Vincent's spirilla, Diplococcus catarrhalis, spirochetae, streptococci, etc. It is almost impossible to get a non-contaminated culture from the end of a tooth without extracting the tooth most carefully under local anesthesia. Some experts may perhaps get a culture from the pulp canal (doubtful). When a culture is required, I always prefer local anesthesia, because I am not so sure of removing a tooth from its socket without contamination, with increased flow of saliva and bleeding of the parts under general anesthesia. The attempt to get a culture by going through the gum and alveolus, and not removing the tooth, seems to be a waste of time, because in most cases it is impossible to get an uncontaminated culture, for the tissue through which one must pass cannot be sterilized.

The status of pulpless teeth has not been settled as yet by the dental profession. My opinion is that *nearly all pulpless teeth* are infected, and that most of them cannot be made safe for the patients that are carrying them. Knowing as we do that most pulpless teeth are *infected*, have we the right to say that because the patient is not having any trouble with them at present, and that the radiographic findings are negative, the teeth can be watched and checked up from time to time, and when they do cause trouble, they can be removed? How much damage will be done by that time? How can we tell when they will begin to cause trouble? How can we measure the amount of absorption that is taking place in one's body from any infection that, at any given moment appears to be giving no trouble? Can we say that this man, a young husky, can carry infected teeth for five years, and that another, who is not so strong, can carry the said streptococcic

teeth for two years only, when they will be removed? I think that this reasoning is dangerous, and I know of many instances where it has proved fatal to the patient.

I am sorry to make the statement, but I feel at *this time* that most pulpless teeth ought to be removed in a patient *fifty years old* (arbitrary age) to reduce, to a minimum, the amount of virulent bacteria and their toxins in his body, just as it is a correct procedure to cut down to a minimum the amount of bacteria in the milk or other food that one consumes. This is, perhaps, rather radical, but it is based upon many years of experience with infected mouths, and is prompted by some very sad experiences in trying to save teeth that the dental surgeon could not *render aseptic*. For how can we say when a tooth is safe or sterile, and who can say when it is giving *trouble* or when it will decide to give trouble from a *systemic* point of view? When we can definitely say that a tooth infection is causing a given systemic symptom, when we shall be able to measure the amount of disturbance that such an infection is giving, then and not until then can we temporize with virulent bacteria in the mouth.

I think it is a poor practice to curet infective alveoli of teeth after extraction. Price divides his cases of periapical infection into *three classes* according to the ability with which the body through its defensive mechanism is enabled to build up a *granuloma* around the apex of an infected tooth. In the first group are those showing considerable evidence of granulomatous formation with rarefaction of the bone, as shown by the radiogram. In the second are those in which there is little rarefaction of bone, and consequently evidence of a poor granuloma, but rather a tendency to condensation of bone in the neighborhood of the infected apex. In the third group are those in which the granuloma and the rarefaction of bone give way to condensation in that position—where the signs of the first group are being replaced by those of the second. Price found, further, that individuals in group one had a very definite immunity to rheumatic disease; in the second group, there existed a definite susceptibility thereto; whereas in the third group, an original immunity gave way to susceptibility. He also found some very interesting facts regarding the biochemistry of the blood in these three groups, and pointed out that the building of a granuloma was a defensive process. Individuals in whom the defen-

sive mechanism was efficient were immune to rheumatism; those in whom it was deficient were susceptible. Some years ago, the great surgeon (and friend of many in this audience) Dr. Joseph A. Blake, asked me not to curet the alveoli after extracting an infected tooth for him. He said, "You know, Dunning, it has taken me many months to build up a barrier around that tooth, and I do not want it broken down at once, so that the 'bugs' at the apex of that tooth can have free access into the walls of the newly formed wound." Ever since this meeting with Dr. Blake, I have refrained from using the curet after extraction of teeth, except in certain more or less rare cases.

The writer believes that it is a dangerous practice ruthlessly to destroy the defensive barrier that nature has built around the apex of a diseased tooth, as it is possible by this procedure to let loose into a patient's system many dangerous bacteria and their toxins. I feel from my talks with Dr. Roseman that he concurs in this opinion. It is a much safer procedure to swab the post-extractive alveoli with equal parts of half strength iodine and glycerine, keep the wound open, and let nature throw off the granuloma or limiting membrane in her own manner "when she gets good and ready to do so." I also believe that it takes a long time for nature to throw off bacteria from an infected post-extraction wound. Many times we do not eliminate bacteria from the jaws by the mere extraction of teeth. Some writer stated that from 20 to 40 per cent of edentulous mouths harbor residual areas of infection. *Infected root-ends and cysts* play an important rôle in the above picture.

I have endeavored to give you my idea of the oral side of the picture of infections that play a part or have a bearing on the subject before us. To say that my knowledge of the diseases of the eye is very limited is to put it very mildly. In the preparation of this paper, I have been greatly helped by Dr. Conrad Berens, who very kindly placed his splendid library at my disposal. The literature has been examined quite carefully and appears to me to be teeming with evidence that infections of the mouth play an important rôle in ophthalmic diseases. I could retail to you many cases reported in the writings of De Schweintz, Berens, Rosenow, Wilmer, Price, Gardner, Bell, McCarltry, Bulson and others, that would go a long way to convince

us that the infections of the mouth have a great influence upon many systemic diseases in general, and also upon the conditions of the eye. It will be sufficient to refer to an article by Rosenow and Mickel, read before the American Society of Clinical Pathologists in June, 1928, in which the authors wrote:

"The influence of dental sepsis on diseases of the eye has been investigated by many workers since Lang, in 1913, published his observation on the subject, and a large number of case reports have been published which demonstrate the value of examination of the teeth in treating diseases of the eye.

"Carefully observed cases of periostitis, orbital abscess, iritis, and uveitis that can be definitely ascribed to dental sepsis have been recognized and described. While the exact process of extension from the teeth to the eyes was in many instances not known, the improvement of the eye conditions that followed correction of the disease about the teeth was considered sufficient evidence that a close relationship exists. When a rather severe chronic corneal ulcer that had failed to yield to common methods of treatment healed readily after the extraction of a diseased tooth, when chronic or recurrent iridocyclitis became quiet following the removal of infected teeth or the treatment of pyorrhea, other probable causes of the disease having been eliminated, the conclusion was justified that the disease of the eye was due to disease about the teeth, or that toxins absorbed from suppurative foci about the teeth contributed to the chronicity of the ocular affection.

"That pyorrhea or partly decayed teeth in the mouths of persons suffering from disorders of the eye do not more frequently appear to have an influence on the course of the disease has been used as an argument against the causal relationship of dental and ocular affections, but if we more fully understood the characteristics of the organisms that produce disease, and the changes in virulency of the organisms and variations in the strength of the resistance which Nature puts up as a barrier to the spread of the disease, we probably could more easily account for the few metastic infections, and arrange a more satisfactory system of prophylaxis.

"*Methods of Transmission.* Eyes, previously healthy, may become diseased by the transmission of organisms from diseased teeth in three ways:

"1. Direct extension through bone. The tooth socket becomes the seat of an infection that destroys the bone of the upper jaw, and through that directly affecting the orbital walls, finally producing an orbital cellulitis or retrobulbar neuritis. Such extensions of the disease process about

the teeth seldom affect the interior of the eye, but are found as abscesses of the lids, particularly the lower, abscesses of the sinuses, emboli, abscesses of the frontal lobe with atrophy of the optic nerves, and periostitis.

"2. Direct extension of the process along the periosteum of the malar bone invading the orbit and giving rise to processes similar to those mentioned.

"3. Transfer of organisms from the focus at the tooth to the eye by the blood stream. This is the most frequent means of transfer of infection from the teeth to the eyes, and consequently is the most important.

"It is often difficult to demonstrate that infections about teeth are responsible for any individual case of eye disease. Extraction of affected teeth does not always yield beneficial results. But it should be remembered in this connection that possibly all sources of organisms responsible for the eye affection were not eradicated with the extraction of the teeth, that organs other than the eye have been invaded and become secondary foci for the production of the same strain of organism, or that even after extraction a septic picket may have remained in the diseased jawbone. Recurrent iritis, keratitis, uveitis, and transient disturbances of ocular motility occur from these secondary foci after the removal of infected teeth and are to be combated by the use of autogenous vaccines.

"Some forms of eye disease that formerly were believed to be due to autotoxemia, injury, blood disease, rheumatism, or were idiopathic, have been demonstrated to be due to the actual presence of bacteria carried to the eye from suppurative foci about the teeth or tonsils. Rosenow has shown that organisms taken from diseased teeth are capable of producing disease of the eye when injected directly into the blood stream of animals; and furthermore that such organisms have a selective affinity for certain structures of the eye, and that within limitations this selective affinity is characteristic of the strain and is maintained through several cultural transplants of the organism. He has demonstrated that organisms grown from the tooth of a person suffering from muscular rheumatism produce iritis in the eyes of animals as well as hemorrhages in the skeletal muscles.

"The barrier that nature erects in defense of the eye may be effective against a specific strain of organism, and since organisms about the teeth multiply in kind as well as in number the fact that an eye becomes diseased in a person whose mouth has harbored pus-producing foci for years indicates that his barrier has been lowered, or it may mean that a new organism has been introduced against which no barrier has been erected.

"Attacks of eye disease produced through dental sepsis may be of any degree of severity. Mild attacks of conjunctivitis or iritis may occur

which amount to no more than a transient hyperemia of the conjunctiva, or a slightly troublesome photophobia. A rapid succession of mild attacks extending over a period of several years has been demonstrated to be due to periapical infection. The iris and choroid are more often attacked by organisms from apical infection although optic neuritis, scleritis, keratitis and conjunctivitis are reported to have been found.

"Since the introduction of a dental section at the Mayo Clinic, the relationship of diseased teeth and lesions of the eye has been carefully studied. Definite relationship has been established in a number of cases of acute inflammatory eye lesions and similar lesions produced in animals by means of methods described by Rosenow. Autogenous vaccines made for the patients have been used to supplement the treatment with highly gratifying results. In many conditions of the eye other than inflammation from infection, the dental examinations have given valuable aid and results which are encouraging and warrant further investigation."

DISCUSSION

Conrad Berens, M.D. (New York City): Everyone must have been impressed by the conservative tone of these interesting and instructive papers. There is no situation in ophthalmological diagnosis where conservatism is more appropriate than in the attempt to evaluate etiologic factors in inflammatory eye diseases. Several questions raised by the essayists may bear re-emphasis. When the ophthalmologist is confronted by an inflammatory eye lesion may he decide definitely that dental infection is a factor? Tuberculosis, syphilis, and gonorrhea must first be excluded. In the case of tuberculosis, this may be exceedingly difficult, since even the delicate Mantoux test may be negative and yet tuberculosis may be present. Having ruled out these three diseases, several other foci of infection are usually found. Unless fixation tests from bacteria of dental origin afford a means of making a specific localizing diagnosis, it is impossible to exclude them as factors. When the sinuses, tonsils, prostate, gall bladder, and other foci can be excluded, the intestines can rarely be positively ruled out. From experience during the past few years it is my impression that the complement-fixation tests give suggestive information, but that these findings are not absolutely infallible. A patient with chronic uveitis whose fixation test with *Streptococcus viridans* of dental origin was four plus last year, and who had four periapical alveolar abscesses, was recently re-examined. Since the foci have been removed the fixation test is no longer positive with dental streptococci, but the uveitis and rheumatism have flared up and there is a four plus fixation with pooled strains of bacteria of intestinal origin.

Having determined the possible dental origin of the inflammatory eye condition, how should these findings be discussed with the patient, and how should we evaluate the reports of the dentist, the specialist in oral diagnosis, and the oral surgeon? In the present state of our knowledge it seems impossible to state positively that the eye lesion is caused by a dental infection, but we may tell the patient that the probable dental infection is the only factor found, or that the teeth are one of several factors. As the dental infection can usually be completely eliminated, which is not always true of several of the other foci of infection, it may be advisable to commence with the teeth. In evaluating the data found by the several oral specialists, it sometimes seems to the ophthalmologist that the tendency of the conscientious surgeon and the dentist who has treated the teeth is toward too great conservatism, and that the oral diagnostician who neither treats patients nor operates is in a position to render the least biased opinion. All these opinions should be carefully weighed in serious cases and negative roentgenographic evidence should not be considered conclusive, especially when pulpless teeth are present. If several teeth are to be extracted, the patient should be warned to have no more than two extractions at one time. One patient, in spite of this advice, had eight teeth extracted at once, and then attended a directors' meeting. He was put to bed with a heart attack the next day, and the iritis which had been mild in one eye became fulminating in both eyes and was complicated by glaucoma. This patient was confined for several months as the result. I am convinced from several unfortunate experiences that Doctor Dunning's rule of not curetting after extraction should be more generally followed.

How far one should go in urging the extraction of teeth in patients whose vision is not seriously endangered is a question that should be left to the judgment of the patient, but he should be warned of the possible danger to his eyes from the dental focus. When the teeth have been extracted, have we any way of determining whether the dental infection was a factor? Rabbits may be injected to see whether the organisms will localize in the eye, as suggested by Rosenow. Although elective localization is not proved, and Doctor Steinbugler has advanced evidence to discount this theory, several observers have apparently confirmed Rosenow's work (Nickel, Neissen, and Nakamura). Benedict (*Trans. Am. Opth. Soc.* 1926) has found this a useful method of selecting bacteria for incorporation in the vaccine, which may be made from each of the organisms found and the patient's intradermal sensitivity tested. This method is more applicable to clinic and office practice than Rosenow's technic of animal inoculation. It has

been studied thoroughly by Thomas, Famulener and Touart (*Arch. Internal Med.* 34:85, July, 1923), and we have used vaccines selected by this method in practice for several years with the following results taken from a few of the cases in which the dental factor seemed important—*total number of cases studied in this series, 54*:

Staphylococcus aureus was found in	34 cultures
Staphylococcus albus was found in	10 cultures
Streptococcus hemolyticus was found in	2 cultures
Streptococcus viridans was found in	36 cultures
Streptococcus communis was found in	17 cultures
Streptococcus mucosus capsulatus was found in	2 cultures
Pneumococcus was found in	2 cultures
Micrococcus catarrhalis was found in	2 cultures

Streptococcus viridans was recovered in pure culture 16 times. Of the 54 patients, 51 apparently improved, 39 with vaccine treatment, and 12 without vaccine treatment. Three patients showed no improvement. In one instance vaccines were used; in the other two no vaccine was used. (See *Table 1.*) The value of these data in regard to the effect of treatment is questionable when one realizes that local and general medical treatment was used, and other foci were also cared for, frequently with the addition of vaccines and filtrates applied or injected locally in cases where chronic sinusitis was a complicating factor. If the patients show marked intradermal hypersensitiveness to the organisms found, it possibly suggests that the dental infection was a factor in the eye disease. When there is no increased hypersensitiveness, the vaccine is usually not given unless the eye lesion is serious or progressing.

The last question raised by Doctor Steinbugler is that of vaccine therapy. We would like to know whether vaccines are of value and if so, what type to use. Vaccines are used after the extraction of one tooth, to raise local and general resistance before other teeth are extracted, provided the general condition of the patient warrants vaccine therapy and marked intradermal hypersensitiveness is proved. If the patient has a chronic inflammatory eye lesion and all the dental sources of infection cannot be eliminated, the vaccine should be given in small doses for a year and may be combined with iron if there is anemia. If stopping the vaccine at the end of a year results in no increase in the local inflammatory condition, it may be discontinued. We should not forget, however, that immunity is of short duration where streptococci are concerned, and, as Steinbugler has stated, I have sometimes thought the treatment was merely non-specific desensitization and not true immunization. However, until this point has been

TABLE 1

Established dental infection concomitant with specific eye diseases

DIAGNOSIS	CASES NO.	ORGANISMS PRESENT	TIMES PRESENT NO.
Cataract	13	Streptococcus viridans	11
		Streptococcus communis	3
		Staphylococcus albus	3
		Staphylococcus aureus	7
Chorioretinitis	11	Streptococcus viridans	6
		Streptococcus communis	4
		Streptococcus mucosus capsulatus	1
		Staphylococcus albus	3
		Staphylococcus aureus	7
		Pneumococci	1
Glaucoma (primary)	1	Streptococci	1
		Staphylococci	1
Glaucoma (secondary)	12	Streptococcus viridans	11
		Streptococcus communis	4
		Streptococcus mucosus capsulatus	1
		Streptococcus hemolyticus	1
		Staphylococcus albus	3
		Staphylococcus aureus	6
		Micrococcus catarrhalis	2
Iritis (acute)	2	Staphylococcus albus	1
		Staphylococcus aureus	2
Keratoiritis	1	Streptococcus viridans	1
		Streptococcus hemolyticus	1
		Streptococcus communis	1
Retrobulbar neuritis	4	Streptococcus viridans	2
		Streptococcus communis	2
		Staphylococcus aureus	2
Thrombosis of central artery	2	Streptococcus viridans	1
		Staphylococcus aureus	2
Uveitis, chronic (with deposits on Descemet's membrane)	8	Streptococcus viridans	4
		Streptococcus communis	4
		Staphylococcus aureus	8
		Pneumococci	1
Total.....	54		

proved, specific vaccines are to be preferred to non-specific ones. It should be borne in mind that vaccines constitute only one method of raising resistance, and may actually do harm if the patient's power of reaction is diminished. All other factors, such as proper diet, sufficient rest, exercise, sunshine, and fresh air should also be considered.

Although it may seem that many eye conditions are improved by extracting teeth, we have seen the same diseases cured by medical treatment alone, or possibly by Nature, for the tendency of almost every disease is to improve. The eye disease can develop only if resistance is lowered, and much absorption of infection can occur from the teeth without producing an eye lesion, provided resistance is adequate. Some authorities still believe that inflammatory eye diseases are not caused by sinus or dental infection. Although I hold the opposing view, and believe that many postoperative complications are also caused by these infections, conclusive proof of either contention is difficult to establish, and a lengthy discussion of this phase of the subject would be out of place here.

I have thought for a long time that some forms of so-called senile cataract, and even so-called primary glaucoma, were sometimes due to the results of chronic dental and other infections acting either directly on the eye tissues or indirectly through disturbance of the nervous, endocrine, or vascular systems, with secondary changes in the blood and nutritive fluids of the eye. In a recent paper, Weeks expressed the same view in regard to the etiology of cataract. I have seldom found a patient with progressing cataract or glaucoma who was not suffering from some chronic infection. In the cases where the infection could be eliminated or resistance raised against the infection, the eye diseases in many instances have apparently been arrested or the progress retarded. It is apparently impossible to prove this theory experimentally, but clinical results must sometimes be considered. Although these points and the differences of opinion should be explained to our patients, it is well to warn them of possible danger from dental infection should resistance become lowered or the eye tissues become sensitized by previous inflammation.

James G. Dwyer, M.D. (*New York City*): I think the conservative position of all the speakers tonight is the most striking and important thing to be emphasized. Medicine goes in cycles. Even in our own time, it has been a swing of the pendulum one way or the other. I think the pendulum has swung too far concerning focal infections, in that one specialist goes for teeth, another for tonsils, another for intestinal tract, and so on. When a patient consults us with some of these infections, we tell him if he gets

all his teeth out and sinuses opened, and tonsils out and gall-bladder opened, and thirty feet of the intestinal tract cleaned out, he might get well. This is meant to be humorous, but it shows how open our minds have to be kept on all of these questions.

The striking things about focal infections in general that have emphasized themselves in my mind are these: we never get them in young people. It is essentially a condition that goes on for years before it manifests itself. Although an apical abscess, or diseased tonsils may be present, these infections, usually do not manifest themselves until middle life. Then we get a sudden flare-up, an iritis or a choroiditis, or an inflammation around a joint. The other point that emphasizes itself is the presence of pus under pressure. I think these are the two cardinal things, based on clinical evidence and also upon some experimental work I did with Dr. Zinsser some years ago, namely, of trying to reproduce focal infections by bacterial sensitization of joints or the eye. To my mind, the mistake made in quoting experiments on *Streptococcus viridans* for instance, from an apical abscess injected into an animal and expecting it to localize in an eye, is the oversight that these infections have lasted in the original host for months, and usually years. Friedberger has found that if you take either of two such organisms as *Bacillus typhosus* or typhoid bacillus, and the basal protein or the anaphylatoxin, as you may call it, of the *Bacillus prodigiosus* that is never pathogenic, you can, by using this protein in a system of injection experiments on animals that can be completed in several weeks, protect them against one or the other organism. In other words, you can immunize an animal with the prototoxin or basal protein from *B. prodigiosus* and thus protect the animal from a lethal dose of the typhoid germ.

These infections are always found in a part of the body where there is a change from the blood supply to the lymph supply. The classical example is that of the eye. The infections occur in the irido-ciliary axis, in the choroid, or around a joint, but not in a joint. Pus does not arise in a joint from focal infection, but an infiltration occurs where there is, anatomically or physiologically speaking, a change from the blood supply to the lymph supply. In such locations an infiltration proceeds continually and the bacterial products, not necessarily the bacteria themselves, probably sensitize these points—in the eye, the irido-ciliary axis. Then what happens? After years—after the eye has been sensitized in one case, or a joint in another—there is a sudden flowing in probably of a few bacteria. It may be sensitized from a tooth, but an intestinal upset may, on this nonspecific basal protein proposition, "light up" the infection. That is why

we have to keep an open mind. We also have to keep in mind, as Dr. Dunning said, the changes in the chemistry of the body due to lack of vitamines, etc.

Dr. Steinbugler spoke of the routes of transit. I don't consider it very important to trace an infection from a tooth up through an antrum or sinus, and then up to the optic nerve, for instance. The direct path usually gives us more of the type of a phlebitis or an edema of the optic nerve, as distinguished from the ordinary type that probably travels through the lymph channels into the blood stream, whether from a tonsil or a tooth. Although the site has probably nothing to do with it, I believe it is a matter of protein sensitization over a period of years, and then suddenly being exacerbated exactly as a person may be sensitive to horse serum for years, and then get a dose of horse serum and "pass out" with anaphylaxis. The sensitizing limit is often inherited, often acquired. Then comes the anaphylactic dose, the poisoning dose, corresponding to the poisoning dose of the bacteria, that causes rheumatism, iridocyclitis, etc.

Some of the work we did along these lines, to illustrate the time element in an infection, was to take protein extracted, for instance, from streptococci or staphylococci, and inject it in one series of rabbits into one joint, in another series of rabbits into another joint, in another series into the anterior chamber of the eye. This was done five or six times with the extracted protein. Then, after four to seven weeks, the animals were given sublethal doses of the corresponding organism. There followed abscess formations in joints in some; in others, in the eye; and so forth. These results parallel those of the so-called elective localization experiments. It is important to consider the period of time over which the focal infection is spread. We never find infections in the tonsil, for instance, of a child. The worst eye infections or the worst focal infections are in men and women who had their tonsils taken out by the old method years ago, when big pieces of tonsil were left in and scar tissue formed. Then the ordinary secretion of the tonsils, instead of getting out of the mouth, forms a closed sac and the pus is pumped in—and a sudden infection "lights up" a focal infection!

Arthur H. Merritt, D.D.S., F.A.A.P., F.A.C.D. (New York City): The subject of mouth infections and their relationships to those of a secondary nature has been covered so comprehensively by Drs. Gillett and Dunning as to leave little more to be said. I shall therefore limit myself to a very brief consideration of two or three phases of the subject by way of emphasis and elaboration. The first of these is infected pulps in *vital* teeth. In the light of more recent investigations, there can be no

doubt that under certain conditions the pulps of vital teeth may become the seats of chronic infections without objective or subjective symptoms. One of the most frequent causes of such infection is dental caries. The deeper the carious process and the younger the patient, the greater is the probability of pulp infection. This is true because of deeper penetration and the greater amount of organic substance in the teeth of young patients. And since dental caries is essentially a disease of childhood and adolescence, pulp infection as a source of metastatic infection should always be considered in a search for possible foci of infection. Another source of pulp infection is periodontal infections as they are represented by gingivitis and periodontoclasia. Noyes has shown that the histopathologic changes occurring in the pericementum and walls of the alveolus, and referred to as rarefying pericementitis fibrosa, are caused by the invasion of these tissues by mouth organisms, which may be deeply embedded in these tissues without causing anything more serious clinically than a mild gingivitis. This being true, it is logical to expect pulp infection as well as periodontal infection in all advanced cases of periodontoclasia. While it is probably true that pulp infection occurs under the above-mentioned conditions, and should always be considered in the search for possible foci, there is no reason why we should extract all suspected teeth. Properly treated by well-known operative procedures, the primary focus in pulp infection, whether it be caries or periodontoclasia, can be eliminated. When this is done, one is justified in assuming that as a rule the secondary focus, as represented by pulp infection, will be taken care of by nature's own defensive forces. This is a feature of the focal-infection problem that is too often overlooked. We have a right to assume that, having removed the primary focus, nature will take care of the secondary focus provided, of course, the tissue is not too seriously damaged and the blood stream has access to it. Immunity is a factor to be considered in all these cases.

The second factor in mouth infections is that of pulpless teeth. This is perhaps the most common dental infection and potentially the most dangerous, a fact emphasized by Dr. Dunning. He is undoubtedly right in saying that these infections usually occur by way of the pulp and periodontal route rather than the blood stream. His references to these teeth as "non-vital," implying thereby that they are "dead," is a little misleading as has been pointed out by Dr. Gillett. His statement that all "non-vital" teeth are infected, and that most of them cannot be safely retained, can probably be explained by the fact that as an oral surgeon he sees only the failures in these cases. Unfortunately, there are many failures in the field owing in part to faulty diagnosis and bad technique. There

are also many successes. But it must be said in all seriousness that every pulpless tooth is open to a measure of suspicion. The only satisfactory solution to this whole problem is through prevention. No treatment can ever be wholly satisfactory.

Still another possible source of infection is the area of periapical infection that remains in a jaw after extraction of a tooth. The removal of an infected tooth does not necessarily remove the infection. One should therefore include, in his examination, a radiographic survey of all edentulous areas with a view to determining so far as that is possible whether residual infections as these are represented by root ends, granulomas, etc., may be present. Here again, one should exercise caution in diagnosis and treatment, for in this field as in some others radicalism has sometimes prevailed. Experience and judgment are of first importance in considering this aspect of oral infections. Dr. Dunning's observations regarding curettment in many of these cases should be seriously considered.

The fourth and last phase of this subject to which attention is called is that of periodontoclasia or so-called pyorrhea. President Davenport and Dr. Gillett have both asked that in my discussion this aspect of the subject be amplified, since it is one that could be touched upon only generally in the papers under discussion. It is probably not too much to say that no infection occurring in the mouth is more frequently overlooked or more worthy of serious consideration. Its chief etiologic factor is *Streptococcus viridans*, which is found in large numbers in all periodontal lesions. Its causative relationship is due to its invasive nature rather than to its specificity, which explains why vaccines are of little or no value in the treatment of periodontoclasia. This is not to say, however, that they may not, in certain cases, be helpful in clearing up the metastatic infections of this disease, and their possible use should always be considered.

After a study of pyorrheal infections undertaken at the Mayo Clinic by Cook and Stafne (16) for the purpose of ascertaining relationships to metastatic infections, they consider that periodontoclasia is quite as much a cause of ill health as the more generally recognized infections of the tonsil. Yet, by a curious inconsistency, we insist upon tonsillectomy, at the same time that we ignore periodontoclasia, which may involve more or less seriously all the teeth in the mouth. It has been Rosenow's (17) observation that cultures taken from pyorrheal pockets show a wider range of localization than those taken from periapical infections. His experiments showed, moreover, that 37 per cent of the animals injected with cultures obtained from periodontal pockets of patients suffering from iritis developed unmistakable signs if iritis. Haden (18) states that there is

a close clinical relationship between oral sepsis and diseases of the uveal tract. Lang (19) found periodontoclasia responsible for infections of the eye in 139 of 215 cases, while De Schweinitz sums up his observations in the statement that there is "no better established etiologic relationship than that between septic foci in dental areas and certain diseases of the eye" (20).

It is not strange that this should be so when it is realized that not less than 13 per cent of persons between the ages of twenty and thirty, and 88 per cent of all patients over fifty, have periodontoclasia in some form (21). When it is further realized that periodontoclasia is usually represented by pockets with raw and ulcerating surfaces, and by an alveolar process that is slowly being destroyed; that these tissues are the seat of a chronic infection, in which the pus and organisms are more or less under pressure; and that these conditions may exist for years with little or no subjective symptoms, it must be obvious that periodontal, as distinguished from periapical infection, is a type that should receive the serious consideration of both physician and dentist. And let it be said for the information of both, that no disease occurring in the mouth is more easily prevented; that if properly treated before too great progress has been made, it can be permanently cured; and that if neglected, as is so often the case, it will not only reach an incurable stage but also for years will continue a chronic focus of infection with all that that implies. Its treatment, which is a purely dental operation, consists in a subgingival curettage of all pockets; the elimination of the overlying gum tissue by surgical procedures in cases too far advanced for treatment by the more conservative and satisfactory operation of curettment; and the establishment and maintenance of a high standard of mouth hygiene and the stimulation of periodontal circulation. In many cases diet has also to be considered, and foods rich in vitamin C and containing an abundance of calcium, should be prescribed. That which should be kept in mind regarding periodontal lesions is that while we have all taken more or less seriously periapical infections, as is proper, we have too often overlooked another type of mouth infection which though less obvious, and possibly on the whole less dangerous as a factor in secondary infections, is nevertheless one that deserves far more consideration by both the physician and dentist than it now receives from either.

References to literature

(16) Cook, T. J. and Stafne, E. C. 1929 Bacteriologic studies of pyorrhea alveolaris, *Dental Cosmos*, lxxi, p. 115.

(17) Rosenow, E. C. 1914 Mouth infection as a source of systemic disease, *Journal of the American Medical Association*, lxiii, p. 2026.

(18) HADEN, R. L. 1928 Dental infection and systemic disease, p. 69. Lea & Febiger, Philadelphia, Pa.
(19) LANG, WM. 1913 Influence of chronic sepsis on eye disease, *Lancet*, i, p. 1368.
(20) DE SCHWEINITZ, G. E. 1920 Dental sepsis and its relation to ocular disorders, *Dental Cosmos*, lxii, p. 565.
(21) BLACK, A. D. 1918 Roentgenographic studies of tissues involved in mouth infections, *Journal of the American Medical Association*, lxxi, p. 1279.

John Oppie McCall, B.A., D.D.S., F.A.A.P., F.A.C.D. (*New York City*): At the outset, I wish to congratulate Dr. Gillett on his very complete and scholarly paper on this important subject. He has epitomized for us the various relationships that make teeth and the mouth interesting to the physician, and which make the rest of the body interesting to the dentist. His statements sum up present-day knowledge so accurately that no opportunity for disagreement presents itself. He has also portrayed, although not as strikingly as their importance warrants, the weak links in the chain of present-day knowledge.

If I were to seek some point on which I might disagree with Dr. Gillett, it would be as to the mechanism by which diseased teeth produce injury to the digestive organs. Dr. Gillett voices the logical and, as far as it goes, entirely correct view that digestion or, shall we say, the health of the digestive organs, suffers when pus and filth accumulated around the teeth are carried into the stomach. Similarly we have been taught that loss of teeth, i.e., diminished or absent masticating power, causes indigestion. Each of these things is true. Yet it has been my observation, confirmed by many cases in practice, that an even more important source of derangement of the digestive organs is the blood- and lymph-borne infection from periapical lesions having no direct connection with the mouth fluids, but in natural dentitions having a high degree of functional efficiency. The improvement following extraction of teeth in such cases, even when this nearly or quite does away with mastication, is rather startling. While this is digressing from the main topic of the evening, I feel that the excursion may be justified as contributing to the general consideration of metastasis. Dr. Gillett catalogues most acceptably the various infectious foci that may be found in the mouth. I would like to amplify certain of his statements, since some of these foci have received too little attention. I refer chiefly to teeth with infected but vital pulps, and to residual infection. In the early days of the study of focal infection, attention was centered on the gross and most readily discovered lesions, namely, the periapical granuloma and the periodontal pus-pocket, with some consideration for impacted teeth. This attention was rightly given; but as a result

of this early emphasis, there has been a tendency to visualize oral focal infection problems as confined to these lesions. I have seen so many cases carried through the stage of removal of pulpless and pyorrhetic teeth with little or no benefit to the patient, and the same cases yielding brilliant results after the subsequent removal of vital teeth with pulps infected by deep caries, or by the removal of residual infection, that I have been forced to adopt a very critical attitude toward such possible foci.

With regard to intra-ocular lesions, experience with my own case has been interesting and convincing. My personal experience with focal infection dates back ten years, at which time, as a result of a partial neurasthenia, with associated disorders including a mild blepharitis, I had had several non-vital teeth extracted. Improvement was marked and I went along for several years without any pronounced symptoms. Four years ago, however, I began to be troubled with recurrent attacks of blepharitis, with occasional so-called styes, and an almost constant conjunctivitis with a tendency to involvement of some of the deeper structures. At this time, also, I was conscious of a lassitude not readily accounted for; I had nine vital teeth which had been deeply filled, but which gave no subjective symptoms and showed no abnormal periapical condition in the radiogram. In spite of the absence of clinical or radiographic evidence of infection, I had these teeth extracted. Improvement both as to the ocular condition and general health was marked and has been lasting. It must not be inferred that I regard all deeply-filled vital teeth as infected, or that I recommend the extraction of all such teeth. At the same time, the diagnostician must have ever in mind the fact that vital teeth may under certain circumstances become foci of infection. Dr. Dunning has reported his conversation on this subject with Dr. Bassler, whose statements on this point I can confirm, at least in part. Several cases under my observation, of teeth whose pulps had lost their vitality, gave cultures of organisms known to have their habitat in the intestinal tract.

The subject of residual infection has interested me for a number of years. During the past two years, in a histologic study of a number of these cases at the New York University College of Dentistry, I have found that a large percentage of cases diagnosed from the radiogram as being infected have given definite evidence of low-grade inflammatory reaction. Dr. Dunning has expressed himself strongly on the harmfulness of periapical lesions as compared with other oral lesions. I agree with him on this point. The direct and inevitable absorption of toxic material from the periapical region into the depth of the marrow of the alveolar bone clearly explains the harm caused by the infected pulpless tooth. On the other

hand, I have observed that patients having periodontal lesions but no pulpless teeth, even when exhibiting advanced so-called pyorrhea, are on the whole in a surprisingly good state of health.

As regards focal infection and diseases of the eye, I have had the good fortune to gain considerable experience working first in conjunction with Dr. F. Park Lewis of Buffalo, where I formerly practised. I should like at this time to pay tribute to Dr. Lewis both as practitioner and investigator. Contact with him was both a source of inspiration and knowledge. Experience with the cases he referred to me for oral diagnosis taught me that the eye is one of the most delicate organs in the body, as regards its response to blood-borne infections. Not only is the eye readily attacked by such infections, but when once involved it does not respond to anything less than complete eradication of foci. This is, of course, true for many other infections of focal origin, but exceptions are frequently found.

Dr. Gillett and Dr. Dunning take opposite positions as to whether pulpless teeth are or are not invariably infected, and as to whether they can ever be made safe for the patient. Dr. Gillett states the position held by a very large number of men in the dental profession. His position is undoubtedly a strong one, if we may judge by the number of persons about us whom we know to be harboring teeth not only pulpless, but even patently infected, and who possess perfect health as gauged by the most critical standards. Even if we call attention to this fact, we must agree with the statements of Dr. Dunning to the effect that we do not actually know whether "all's well," in the phrase of the mariner, or what the morrow will bring to these people. My experience leads me to side with Dr. Dunning in his recommendation that all pulpless teeth be extracted, as the only safe course to pursue in the light of present knowledge. For example, I see many people who have pulpless teeth in their mouths, who claim to be in good health, but whom I regard as being far from that state of high health which they claim. These people suffer from headaches, indigestion, neuritis, neuralgia, drowsiness, lassitude, and a number of ailments not always brought out in a routine physical examination. Such people should have no pulpless teeth in their mouths. But for those hardier ones who seem to defy the rules of the game with impunity, we have to fall back on generalities. For these people we shall have to await the development of more delicate tests than those now available for determining the state of their resistance. Is it possible that the eye itself, because of the very delicacy of its highly developed tissues, may furnish a means for making such a determination?

PHYSICO-CHEMICAL STUDIES ON THE STRUCTURE OF DENTAL ENAMEL

III. A Method for Determining the Rate at Which a Salt May Penetrate the Enamel[1]

HENRY KLEIN, D.D.S., Sc.D.

Department of Chemical Hygiene, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Maryland

CONTENTS

I. Introduction 727
II. Experimental 727
III. Results 729
IV. Discussion 730
V. Summary 731
VI. References to literature 731

I. INTRODUCTION

In previous communications (1, 2) it has been shown that the enamel of dog canine teeth is permeable to water and salts, and that the permeability may be evaluated in terms of a measure of permeability designated the Co.P value. The present communication is concerned with a study of the rate at which a salt may penetrate the enamel.

II. EXPERIMENTAL

Dog canines were extracted carefully without injury to the enamel. The pulp was removed, and the pulp chamber cleansed of all organic debris. The teeth were sawed through at the cemento-enamel junction, and the root portions discarded. The crown was then placed in distilled water, which was changed daily for from one to two months, to wash out salts and other soluble substances in or on the structure.

[1] The experiments here reported were carried out under the direction of Dr. W. R. Amberson, at the Department of Physiology, Medical School, University of Pennsylvania.

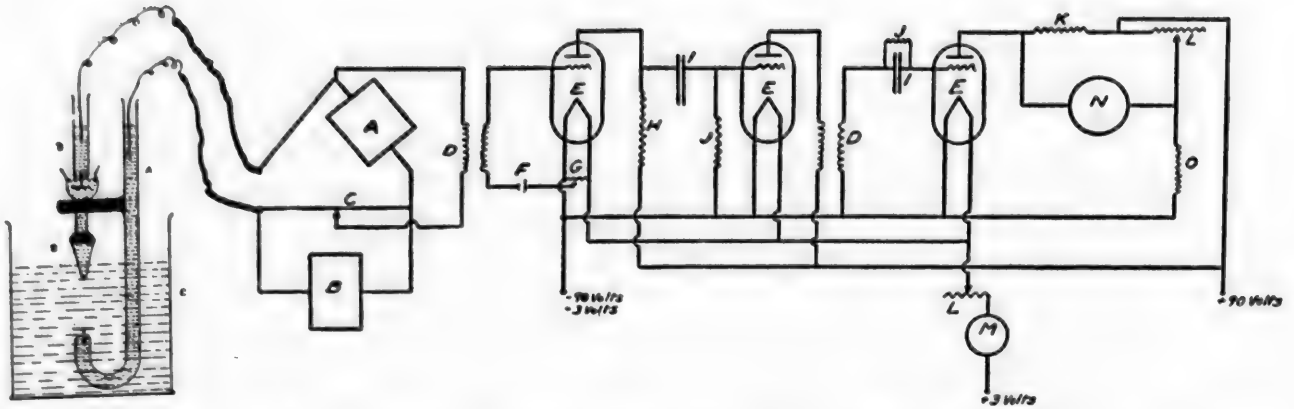


FIG. 1. DIAGRAM OF ELECTRICAL HOOK-UP FOR MEASURING CONDUCTIVITY OF A TOOTH, USING VACUUM-TUBE AMPLIFIER

A—variable resistance. *B*—audio-oscillator. *C*—slide-wire resistance. *D*—audio-frequency transformer. *E*—vacuum tube. *F*—grid battery. *G*—potentiometer. *H*—150,000 ohm resistance. *I*—0.01 M.F. condenser. *J*—5 megohm resistance. *K*—25 ohm resistance. *L*—25 ohm variable resistance. *M*—ammeter. *N*—galvanometer. *O*—12,000 ohm resistance.

To determine the velocity at which a salt moved into enamel, the conductivity method developed by Bronk (3) was used.

The apparatus consists of a Wheatstone bridge, using three known resistances, two of which are variable, and an unknown resistance, which in the experiment is the resistance of the tooth to a 1000-cycle oscillating current. The unbalance of the bridge is followed with a vacuum-tube amplifier instead of the usual telephones. The initial resistances of the teeth are so high that the use of telephones is contraindicated for null-point determinations. *Fig. 1* illustrates the set-up of the apparatus.

After one to two months' immersion in distilled water, changed daily, the tooth is removed, dryed with filter paper, and set up in the apparatus (in the position shown in *fig. 1*). The tooth is sealed to a small tube whose upper end is enlarged to receive one of the platinum electrodes (*B*). The pulp chamber of the tooth, and the glass tube to which the tooth is attached, are filled with pure mercury. The tip of the tooth is then immersed in 0.1 *M* KCl in the beaker (*C*). A second platinum electrode (*A*) is also immersed in this salt solution.

The osmotic gradient that exists across the tooth structure consists of a fluid, having a very low electrolyte content, in the channels of the tooth, against 0.1 *M* KCl in the beaker outside of the tip of the tooth. A reading of the resistance of the tooth structure to the oscillating current, which is furnished by the audio-oscillator (*A*), is made immediately after the tip of the tooth is immersed. An oscillating current is used because such a current does not contribute to the transport of salt across the structure. The tip of the tooth is allowed to remain in contact with the salt solution for forty-eight hours. During this time readings on the resistance are made at frequent intervals.

III. RESULTS

When the tooth is arranged in the apparatus as described above, and resistance readings are made, the resistance of the tooth falls gradually from an initial reading of approximately 900,000 ohms to about 5,000 ohms. The rate of fall of resistance appears to be dependent upon the osmotic gradient existing across the structure. When a salt ten times more concentrated is placed against the tip of the tooth,

after all salts have been washed out again by a two months' treatment in distilled water changed daily, the rate of resistance-fall is much more rapid, indicating a more rapid penetration of salt into the tooth structure. These relationships are shown in *fig. 2*.

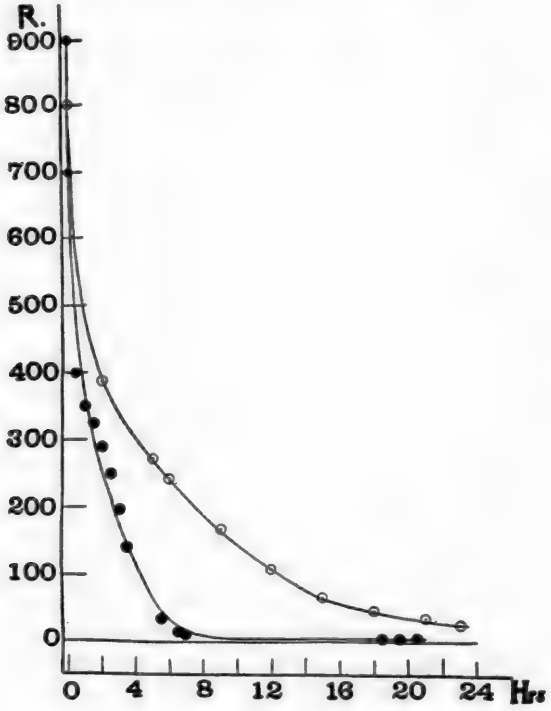


FIG. 2. GRAPH SHOWING DIFFERENCES IN RATE OF SALT PENETRATION, DETERMINED BY MEASUREMENTS OF RESISTANCE

Fall in resistance: open circle with tip of tooth in 0.1 *M* KCl. Fall in resistance: closed circle with tip of tooth in 1.0 *M* KCl. Ordinates: resistance in ohms times 1000. Abscissae: time in hours.

IV. DISCUSSION

When the coronal portion of a dog canine is immersed in distilled water for from one to two months, so that all salts and other soluble substances present in or on the structure can be washed out, one would expect that the resistance readings would be high. Since the con-

ductivity of distilled water is low, a structure washed for a relatively long period in distilled water may be expected to show a low conductivity. Such is the case experimentally. The initial high resistance readings indicate that soluble substances can be washed out of a tooth, and that distilled water is able to penetrate to such an extent as to render the structure exceedingly resistant to the passage of an electrical current. When a tooth is arranged so that opportunity is afforded for salts to penetrate, we expect that, if the salts enter, a series of readings on resistance would indicate the rate of penetration. If tooth structure, including enamel, were impermeable to salts, we should expect very little or no change in the conductivity. If, however, a salt penetrates, then we should expect that, as it enters the channels in the tooth (these channels containing a substance of low electrolyte content), there would be an initial high-resistance reading; and that, as the salt continues to penetrate, the resistance would decrease rapidly, if it entered through numerous cracks and fissures; slowly, if the entry were through small channels. *Experimentally the resistance drops slowly.* The rate of fall is relatively slow, indicating that the salt passes through very small channels in response to the existing osmotic gradient, thus increasing the electrolyte content of the channels and decreasing the resistance to the oscillating current.

V. SUMMARY

(1). An electrical method is presented by which conductivity changes in a tooth may be followed.

(2). The rate of penetration of salt may be studied by this method.

(3). The velocity at which a salt penetrates the enamel is shown to be dependent upon the existing osmotic gradient.

(4). The penetration of salt into the enamel of a dog canine is relatively slow, requiring eight hours with 1.0 *M* KCl solution; twenty-four hours with 0.1 *M* KCl solution.

VI. REFERENCES TO LITERATURE

(1) KLEIN, H., AND AMBERSON, W. R. 1928 *Journal of Dental Research*, ix, p. 667.
(2) KLEIN, H. 1930 *Ibid.*, p. 447.
(3) BRONK, D. 1926 *American Journal of Physiology*, lxxvi, p. 179.

THE EFFECTS OF STRONTIUM ADMINISTRATION ON THE HISTOLOGICAL STRUCTURE OF THE TEETH OF RATS

HENRY KLEIN, D.D.S., Sc.D.,[1] J. ERNESTINE BECKER, B.S., M.A., AND E. V. McCOLLUM, PH.D., Sc.D.

Department of Chemical Hygiene, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Md.

CONTENTS

I. Introduction 733
II. Experimental 735
III. Results 738
IV. Conclusion 738
V. References to literature 738

I. INTRODUCTION

The effects upon the animal organism of the feeding of strontium salts has been studied by a number of workers. Stoeltzner (1908) and Lehnerdt (1910) have reported that the feeding of strontium salts results in the production of excessive amounts of osteoid tissue in the bones. Shipley, Park, McCollum, Simmonds, and Kinney (1922) reported similar findings. They used McCollum's diet 2581 as a basal one, which is composed as follows:

Diet 2581	*Percent*
Cooked, dried beef liver	20.0
Casein	10.0
KCl	1.0
NaCl	1.0
Dextrin	65.0
Butter fat	3.0

This diet is deficient in calcium. When calcium was added, rats developed in a normal manner. Without an addition of calcium, little

[1] Research Fellow supported by grants from the Research Commission of the American Dental Association, and the Dental Staff of the Johns Hopkins Hospital.

growth was possible. In order to determine the effect of strontium, diet 2581 was modified as shown below:

Diet 2581 + strontium	*Percent*
Cooked, dried beef liver	20.0
Casein	10.0
KCl	1.0
NaCl	1.0
$SrCO_3$	2.2
Dextrin	62.8
Butter fat	3.0

It will be noted that this diet is almost identical with low-calcium diet 2581, except that strontium carbonate equivalent molecularly to 0.6 percent of the element calcium has been added.

As mentioned above, Shipley and co-workers found that, as a result of feeding diet 2581 without an addition of either calcium or strontium, young rats did not grow well. With the optimum amount of calcium (0.64 percent) they grew normally. Without calcium, but with strontium added (equivalent to 0.6 percent of calcium), they were able to grow fairly well. Cod liver oil added to the strontium diet assisted in the growth. However, even though the strontium and cod-liver-oil additions to diet 2581 improved the growth of young rats, neither was effective in preventing abnormalities in the histological structure of the bones. The most pronounced effect of the feeding of this ration was the production in the skeleton of excessive amounts of osteoid tissue, resulting in a pathological condition similar to that of clinical rickets. The authors concluded that, although strontium is stored in the bone, it cannot be made to serve the function of the calcium salts. In a later paper Kinney and McCollum (4) reported that strontium is deposited in the bones in appreciable quantities.

The demonstration of such changes in the structure of the bones of rats on strontium diets directs attention to the possible effects on the teeth of the experimental animals. Indeed, that an effect on the teeth was observed is noted in the Shipley paper, the authors having stated that "the skulls were very soft and the teeth slender, white, and easily fractured."

Unfortunately the heads of the experimental rats used by the above mentioned workers were not available when the present authors renewed their interest in the possible effect on the teeth of the stron-

tium-fed rats. There were available, however, in the rat colony, a number of animals that had been subjected to strontium diets intermittently through several generations. Examination of these individuals yielded the findings reported below.

II. EXPERIMENTAL

The rats examined were the offspring of a female which had been fed adequate diets with and without strontium, alternately. During gestation and lactation she was fed an adequate diet, 211, consisting of

Diet 211	*Percent*
Wheat	67.5
Casein	15.0
Whole milk powder	10.0[2]
$CaCO_3$	1.5
NaCl	1.0
Butter fat	5.0

The strontium diet, 4598, consisted of

Diet 4598	*Percent*
Cooked dried beef liver	20.0
Casein	10.0
NaCl	1.0
KCl	1.0
$SrCO_3$	2.2
Dextrin	62.8
Cod liver oil	3.0

The latter diet is the same as that used by Shipley and co-workers. The offspring at weaning were given diet 4598 and diet 211, alternately, on the following schedule:

Diet	*Days on diet*
4598	21
211	18
4598	32
211	3
4598	42
211	11
4598	21
211	41
4598	26
211	9
4598	26

[2] Klim, made by the Merrill-Soule Company, was used in this ration.

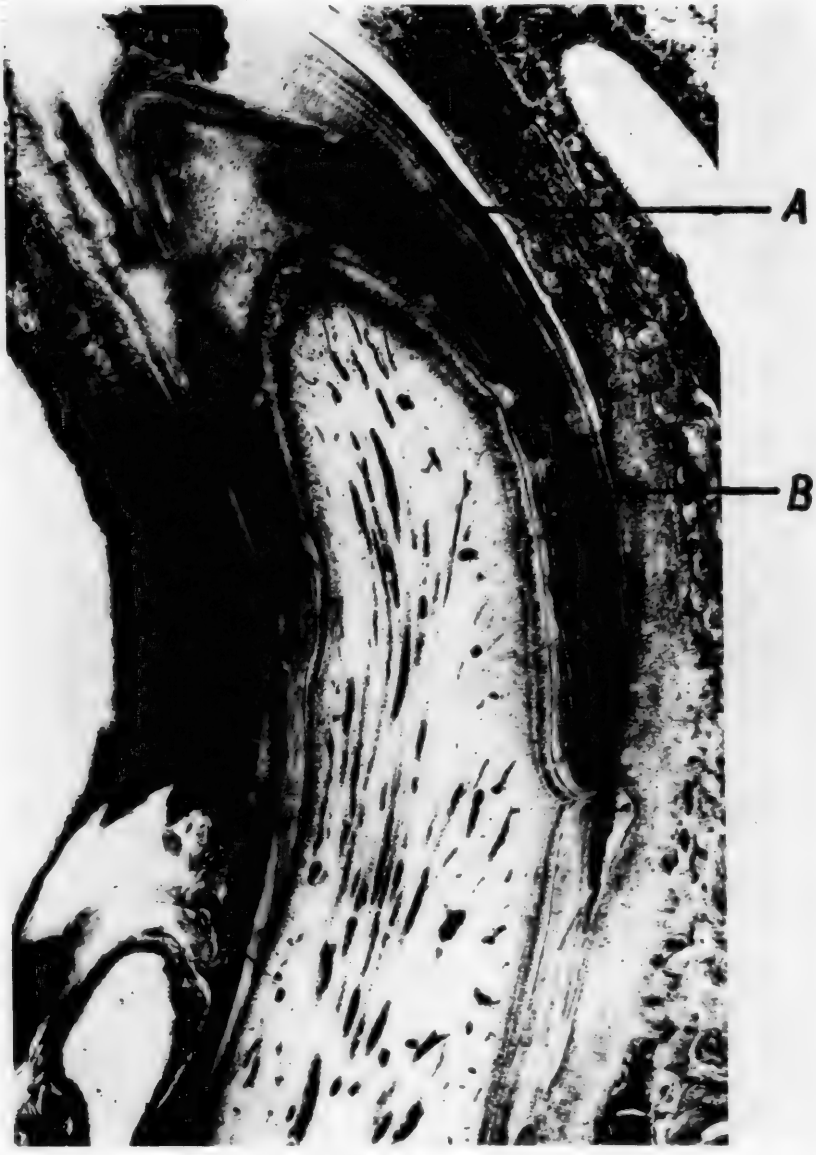


FIG. 1. McCOLLUM RAT NUMBER 232. ALTERNATE FEEDING OF LOW-CALCIUM DIET PLUS STRONTIUM-ADEQUATE DIET. (INCISOR)

A—lamellae of good-poor deposition. B—lamellae of good-poor deposition

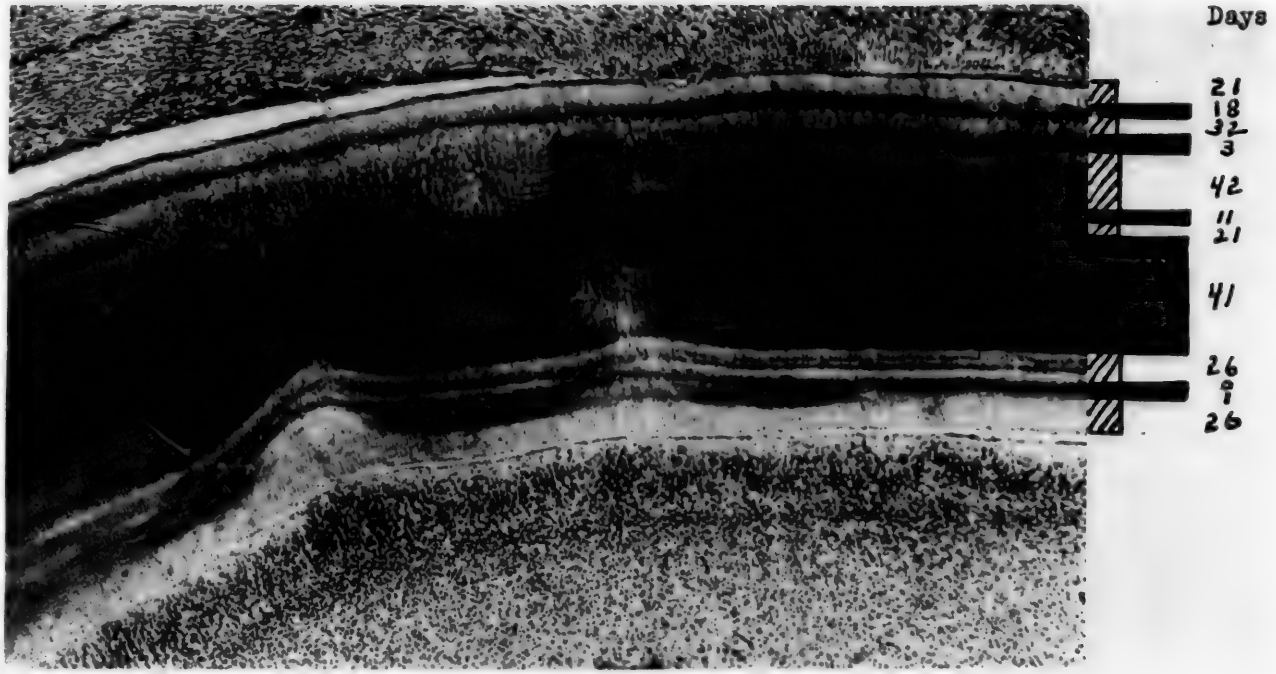


FIG. 2. MCCOLLUM RAT NUMBER 232. ALTERNATE FEEDING. (INCISOR)
▨ Diet 4598—strontium. ■ Diet 211—adequate

At the end of the last period, on diet 4598, the rats were killed and the heads prepared for histological examination.

III. RESULTS

The results of the procedure just described throw light upon the influence of strontium on the calcification processes in tooth development. The effects were clearly shown when the jaws were examined histologically.

When an adequate diet was fed there developed a lamina of well calcified tooth substance. This was followed, during the feeding of the strontium diet, by a lighter lamina of poorly calcified structure (*figs. 1* and *2*).[3] It will be noted, in *fig. 2*, that the different widths of the laminae are such as to suggest that the rate of the calcification processes in the dentine is variable. For instance, the width of the bands during the feeding of adequate diets (these are the more thoroughly calcified bands) do not vary in proportion to the number of days on the diet except in a rather rough way. This may be due also to the fact that the section is not a central one. In general, however, the number of deficiently calcified bands coincide with the number of periods on the strontium diet. The same coincidence of bands and diets may be noted in the more thoroughly calcified bands.

The identification of the bands with the corresponding diets is determined by the fact that the first and last bands, which are poorly calcified, correspond to the ingestion of the strontium diet at the beginning and at the end of the experimental feedings.

IV. CONCLUSION

The bivalent element strontium, supplied in a diet adequate in all respects except in content of calcium, and containing an amount of strontium equivalent molecularly to 0.6 per cent of calcium, produces in the teeth of animals so fed a proliferation of dentinoid. These effects on teeth are similar to the described effects on bone; in both cases strontium in the diet results in a proliferation of uncalcified ground-substance.

V. REFERENCES TO LITERATURE

(1) STOELTZNER 1908 *Bioch. Ztsch.*, xii, p. 119
(2) LEHNERDT 1910 *Beiträge z. path. Anat. und allgem. Path.*, xlvii, p. 215.
(3) SHIPLEY, PARK, MCCOLLUM, SIMMONDS, AND KINNEY 1922 *Johns Hopkins Hosp. Bull.*, xxxiii, p. 216.
(4) KINNEY AND MCCOLLUM 1923 *Journ. Pharm. and Exp. Therap.*, xxi, p. 165.

[3] Sections were prepared with the aid of a Spencer microtome given for this work by Dr. Samuel Lewis and others, of Detroit, Michigan.

CYSTS IN THE INCISOR CANAL

Further Studies of Pathologic Conditions in the Region of the Naso-palatine (Incisor) Canal and Papilla Palatina[1]

JOSEPH SCHROFF, M.D., D.D.S.

Department of Oral Pathology, School of Dental and Oral Surgery, Columbia University, and Dental Department, Mt. Sinai Hospital, New York City

CONTENTS

I. Introduction 739
II. Anatomy of the incisor canal 741
III. Papilla palatina 741
IV. Radiographic appearance of the upper incisor region 741
V. Pathologic conditions of the upper incisor region 742
VI. Case reports 745
VII. Comment 755
VIII. Discussions and conclusions 760
IX. Summary of conclusions 761
X. References to literature 762

I. INTRODUCTION

In a previous article, "Unusual Cysts of the Maxilla" (1), I first called attention in American dental literature to the cyst found in the incisor canal of the maxilla between the central incisor teeth. This cyst has no connection with the teeth, but is derived from the naso-palatine duct or its remnants, occasionally present in humans. These cases have been seen, by many other observers, in radiographic examinations of the incisor region of the maxilla, but were incorrectly interpreted.[2] The larger areas of decalcification were usually considered to be due to dental causes, whereas the smaller circumscribed areas of decalcification were invariably thought to be enlarged incisor foramina. In fact some writers (2) have published warnings against the

[1] Read at a meeting of the William Jarvie Society for Dental Research, School of Dental and Oral Surgery, Columbia University, May 3, 1930.

[2] See Pollia: Fundamental principles of alveolar-dental radiology, p. 253, fig. 155 (1930); Dental Items Publishing Co., Brooklyn, N. Y.

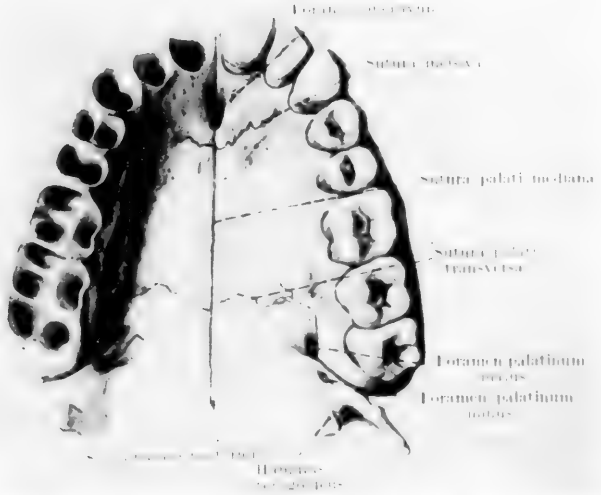


FIG. 1. Naso-palatine (anterior palatine or incisor) canal. (From Sicher-Tandler: Anatomie für Zahnartz.)

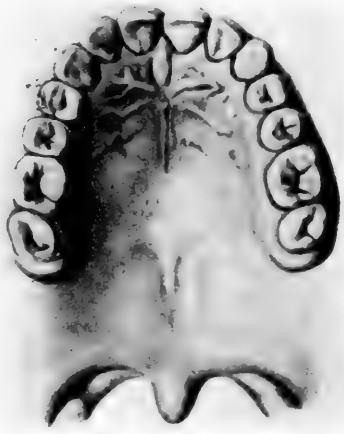


FIG. 2. Papilla palatina and rugae of hard palate.

error of regarding these "enlarged incisor foramina" as *cysts*. The knowledge of this condition being important, practically as well as theoretically, further studies of the naso-palatine canal and papilla-palatina region were made during the past year. In the course of this study several other cases were observed, which will be described in detail. For a better understanding of these cases it is necessary first to review the anatomy of this region.

II. ANATOMY OF THE INCISOR CANAL

Approximately 2 cm. dorsal to the inner margin of the nostril, and in juxtaposition to the nasal septum, the floor of each nasal fossa presents a slight depression. This depression leads into a small canal, the naso-palatine (incisivus) canal, which is lined with mucosa continuous with that lining the inferior nasal meatus. Each canal courses obliquely forward, and with its fellow of the opposite fossa converges toward the nasal septum, descends almost vertically, and passes through the Y-shaped incisor foramen (anterior palatine canal) in the hard palate (*fig. 1*). The canal terminates in the roof of the mouth as a minute slit at the end of the incisive pad or papilla palatina. "Although the two tubes of mucous membrane may join to form a single incisive canal, they usually retain their independence. They are often closed and impervious; sometimes, however, even in the adult, communication is retained between the nasal and oral cavities" (3).

III. PAPILLA PALATINA

On the mucous membrane of the hard palate on the median line, slightly behind the central incisors, is found a spindle or pear-shaped smooth swelling, approximately 2 mm. wide and 3–4 mm. long, called the papilla palatina, which covers the mouth of the incisor canal. On either side of the papilla are the rugae of the palate (*fig. 2*). "Often pin-head-size openings situated near the median line can be seen, which lead into a short epitheliated tube, the remains of the naso-palatine duct" (4).

IV. RADIOGRAPHIC APPEARANCE OF THE UPPER INCISOR REGION

A study of one hundred radiograms of the upper incisor region revealed a wide variation in the appearance of the bone in the area of

the apices of the central incisors. On some films no evidence of the incisor foramen is apparent, while in others it either appears faintly or as a distinct oval or round outline, the area within manifesting different degrees of decalcification. The diameter of these incisor foramina varies, so that it is not always possible to determine by radiographs whether one deals with a normal or a pathologic condition. All of the radiograms studied were taken at the Dental Clinic, in the School of Dental and Oral Surgery, by Mr. Mulhousen, the x-ray technician (*fig. 3*).

V. PATHOLOGIC CONDITIONS OF THE UPPER INCISOR REGION

Inflammatory conditions of the palate localized to the papilla have been observed at some time or other by almost every dentist. Usually the complaints related to this region are so slight, or the symptoms subside so quickly, that very little attention has been given to them. Clinically, the pathology of this region has been observed in the following two forms:

(1) *The simple inflammatory type.* Here the papilla is acutely or chronically inflamed. In the *acute* form, the papilla is intensely reddened and enlarged, and on palpation, soft and puffy in consistency. In the *chronic* form, the papilla has a slight red color, and on palpation a firmer consistency. The pathology involves the papilla mainly.

(2) *The more complex or secretory type.* Here the papilla is slightly redder than normal, but somewhat tumor-like in appearance. On palpation this mass gives the sensation of being under elastic tension. Probing into the openings of the papilla with a very fine probe, one can at times enter these openings for a distance of several millimeters, when a watery secretion flows out alongside the probe. The pathology involves the incisor canal mainly.

Subjectively, slight tenderness or pain is present in the first group. In the second group, in addition to this pain or tenderness, the patient feels a tumor mass with the tongue. Such patients often give the history that the mass varied in size at different times; also that occasionally a salty fluid could be expressed, or sucked out when suction was applied to this region with the tongue.

Etiology. The etiology of the *first* group of conditions is usually simple and related to (1) *trauma*, resulting from the entrance of a

FIG. 3. Some variations in the x-ray appearance of the upper incisor region.

foreign body into the papilla, such as a small fish bone, tooth brush bristle, or a splinter of a tooth pick; or (2) *irritation* of the papilla, during mastication, from an ill-fitting partial or full rubber denture, or poorly constructed fixed bridge pressing upon the papilla. Such a case is cited by Euler (5). The etiology of the *second* group is more complex in that the contents of the naso-palatine (incisor) canal are involved. This condition may be brought about by (1) extension from an inflammation of the papilla upwards into the canal contents, and (2) of dental or (3) nasal origin. (1) Under this subdivison we may have an extension of the inflammatory process of the papilla, caused by the factors mentioned previously, or from a diffuse form of inflammation of the palate such as a stomatitis. (2) As *dental* factors for inflammation of the canal contents, Euler mentions the possibility of an apical infection of the central incisors spreading in the direction of the naso-palatine canal and involving the canal contents, and he has cited one such case. Hauenstein (6), in discussing Euler's paper, maintains that this same condition can also be induced by an extension from a paradentosis of the central incisor teeth, and claims that he has seen several such cases. Hauenstein also cites one case (3) resulting from a *nasal* infection, in which the patient, convalescing from a severe grippe with catarrhal symptoms, presented with painful inflammatory swelling in the region of the papilla. While examining the swelling a serous, sticky, purulent fluid came from the opening in the papilla, suggesting paradentosis. But as the gums and teeth seemed normal, and radiograms showed no signs of pyorrhea or periapical infection, he concluded it was an infection of the naso-palatine canal of nasal origin.

The end result of the inflammation of the naso-palatine-canal contents is the same, whether brought about by extension from the papilla, or from a dental or nasal infection. The exits or slit-like openings of the canal in the palate are occluded, and the inflammatory secretions are confined within a more or less closed space. The pressure of the accumulating secretions is manifested by the tumor formation in the region of the papilla. When the occlusion of the normal openings is temporary, or intervention is affected by simple probing into the mass, or when suction by the tongue against the mass is sufficient to form an exit for the secretions, the swelling may disappear

after a time. However, unless the etiologic factors are eliminated, the swelling recurs. On the other hand, when permanent occlusion of the normal openings into the palate occurs, as a result of the inflammatory process, the accumulating secretions give rise to the retention type of cyst in the naso-palatine canal.

VI. CASE REPORTS

During the past few years it has been my good fortune to study several cases exemplifying the pathology of this region. These cases will now be described in detail.

FIG. 4. Radiograms of incisor teeth and incisor foramen of Case I.

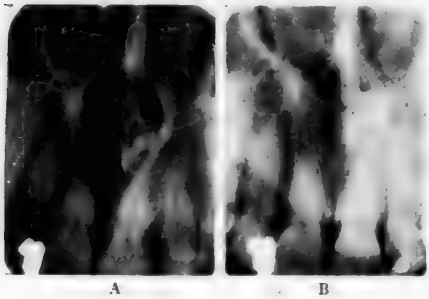
Case I

Miss H. S., age 23, patient of Dr. Wm. Henderson. Six months previously, while in Germany, she had soft swelling on palate slightly posterior to upper incisor region, which disappeared under treatment. Three weeks ago, swelling reappeared and was treated by her dentist, who pierced tumor with sharp instrument. Patient was brought to me by brother of dentist, who knew of my interest in these cases. Examination of mouth, April 29, 1929, showed that all teeth in maxilla were present and in good condition. On palate, posterior to central incisors, there was small depression in region of papilla where, according to dentist who had been treating her, previous swelling had been located. Condition of rest of palate and mouth normal. Radiograph of upper incisor region revealed normal appearance of bone about apices, and fairly distinct outline of incisor foramen (*fig. 4*). Patient was seen again nine months later. During this interval there has been no recurrence of swelling. Radiograph showed no change in appearance or size of outline of incisor foramen.

Case II

History. Miss A. B., age 23, referred by Dr. H. A. Bartels. Four months ago, Sept. 1928, she first noticed soft tumor on palate posterior to central incisor teeth. Swelling disappeared in few days. On Jan. 17, 1929, swelling reappeared and was growing larger. No pain or soreness. Exam-

FIG. 5. Plaster cast of Case II.



A B

FIG. 6. Radiogram of Case II at operation (A), and at follow-up one year later (B).

ination of palate revealed small round bluish-red tumor in region of papilla palatina. On palpation, sensation of tense cystic fluctuation was felt. All teeth were present, normal in appearance, and responded to various tests for vitality. Impression taken (*fig. 5*). Radiograph of upper incisor teeth revealed outline of definitely circumscribed large area of decalcification (*fig. 6*).

Operation. Jan. 27, 1929, under local anesthesia, curved incision pos-

terior to necks of incisor teeth was made on palate. Muco-periosteum was carefully elevated and stripped from tumor adherent to it in papilla region, revealing cyst in mid-line of palate bone in precise site of incisor foramen. Membrane of this cyst was thin and easily broken. Thin, dirty, brownish-white viscid fluid escaped. After stripping out entire membrane, large cavity about 12 x 12 mm. extending upward to the nose was seen. Muco-periosteum was replaced and sutured. Healing uneventful. Radiographs, taken 3/20/29, 5/5/29, and 3/1/30, show bone filling in cavity. Patient has had no subsequent symptoms.

Pathologic report. Tissue composed of fibrous connective-tissue matrix in center of which is cavity lined by layer of squamous epithelium. Some fat cells are seen at one end of section. Debris and some epithelial cells are found within cyst cavity (*figs. 7* and *8*).

Comment. Immediately after operation patient received letter from father requesting her not to undergo operation, because he himself had similar condition and operation had not been resorted to. On communicating with her father, Dr. R. B., following interesting history was received:

"Sometime between the ages of 20 and 30 I noticed several times soft swellings in the front part of my hard palate that eventually discharged in the mouth from a papilla on the median line just back of the incisor teeth. I never examined the discharge carefully or if I did, I have completely forgotten its appearance. This cyst, if it can be termed that, reformed several times while I was from 30 to 40 years old. At about the 45th year, the swelling was repeated, but grew more sensitive and painful and failed to discharge. On consulting my dentist he lanced the swelling, securing a bloody, pus-like discharge. The swelling immediately subsided and healed, and has not returned since. When I asked my dentist if he had ever observed such swellings in other patients he said he had had a similar experience himself, but did not regard it as common."

Have been unable to secure radiogram of this case.

Case III

History. Mrs. L. J., age 49. Four years ago had all upper teeth removed; has been wearing full denture. About five months ago, denture became loose in front due to swelling on palate. This swelling grew larger. Patient went to dentist in Los Angeles, Cal., who treated swelling with ultra-violet rays; tumor burst, discharging watery fluid. About three weeks ago swelling again appeared, and again displaced denture. Patient was referred to me by Dr. I. Rothstein. Examination of mouth, Oct. 29, 1929, showed soft tumor mass on palate on mid-line in region of papilla. Tissue was inflamed, and felt soft and cystic; impression taken (*fig. 9*). Radiograph of area showed large circumscribed area of decalcification symmetrically situated in mid-line of palate in region of incisor foramen (*fig. 10*).

FIG. 7. Photomicrograph of cross section of tissue; low power; Case II. See fig. 8.

FIG. 8. Photomicrograph; high power; Case II. See fig. 7.

Operation. Under novocaine anesthesia, curved incision made along anterior border of tumor, and muco-periosteum elevated carefully from

FIG. 9. Plaster cast of Case III.

FIG. 10. Radiogram of Case III.

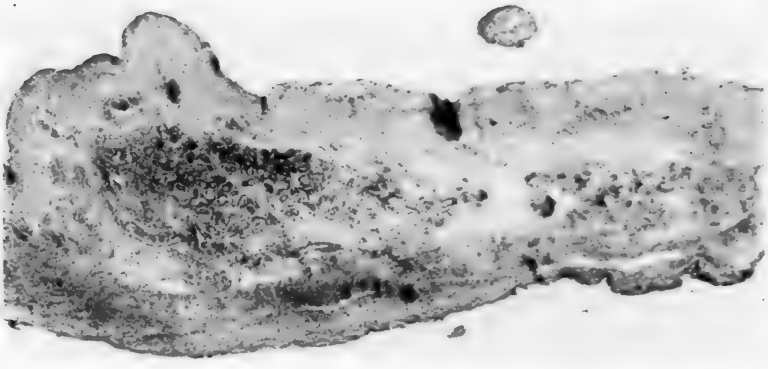


FIG. 11. Photomicrograph of a portion of the longitudinal section of cyst membrane; low power; Case III. See fig. 12.

tumor underneath, which appeared to be cyst; was situated in incisor foramen, which was very much enlarged. Entire cyst was enucleated from

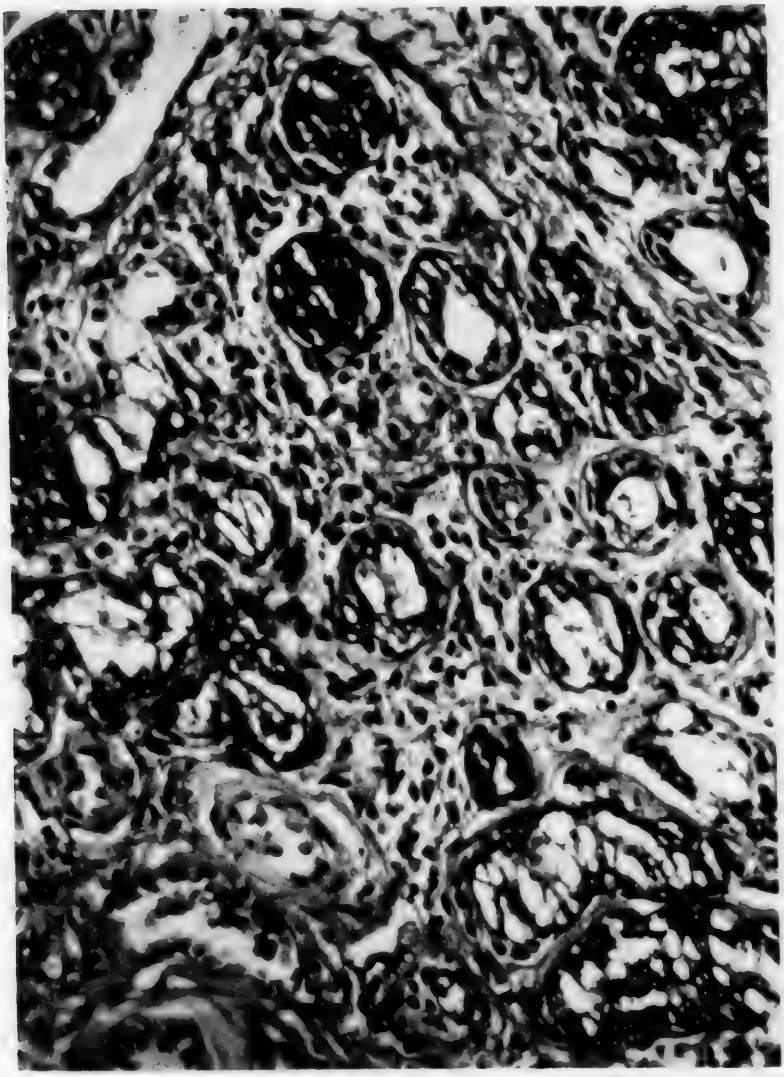


FIG. 12. Photomicrograph; high power; Case III. See fig. 11.

bed, but in this process membrane broke and yellowish watery fluid escaped. Bony cavity was about 15 x 12 mm., extending upward toward nose. Flap replaced and sutured. Healing uneventful.

Microscopic examination of membrane. Longitudinal cross-section of cyst membrane shows tissue composed of fairly thick wall of fibrous connective tissue. On outside of this fibrous tissue traces of thin epithelium layer are seen in some parts. Cyst cavity itself has suggestion of epithelial lining, but no definite epithelial cells are found. Within wall of main fibrous connective tissue, about middle level of cyst membrane, there is nest of epithelial cells and numerous mucous glands are seen. (*Figs. 11* and *12*)

Case IV

History. Mr. S. W., age 54, Mt. Sinai Dental Clinic. Patient edentulous, but before denture was to be constructed radiograph was taken of maxilla, revealing presence of dentigerous cyst containing cuspid tooth in left central cuspid area superimposed on well defined shadow of incisor foramen. (*Fig. 13.*)

Operation. In usual manner cyst membrane was removed and with it came adherent soft mass that occupied incisor foramen (*fig. 14*). Pressure of dentigerous cyst had eroded bony covering of incisor canal.

Case V

History. Mrs. S. H. P., age 30; complained of swelling and pain in upper incisor region, with neuralgic pains radiating to frontal region of head. Her dentist localized pain in left central incisor and, suspecting pulp to be gangrenous, opened into tooth but found it vital. Patient was referred to me. On further questioning, she indicated that occasionally there was fullness on palate near papilla over period of several years, and that she would press on this area when she wanted relief; left central was then tender. Examination of mouth, March 27, 1930, revealed that papilla was swollen and reddened. Very thin probe could be inserted 8 mm. upwards along mesial side of left central. On buccal surface of alveolar process between central incisors, and extending somewhat to left, there was definite round swelling, which fluctuated on palpation and was somewhat tender. Radiogram showed area of decalcification in incisor-canal region (*fig. 15*).

Operation. Curved buccal incision over incisor area was made. When flap was reflected, buccal bone between central incisors was missing, but mass of soft tissue resembling granuloma was noticed. This tissue was removed, which left depression and disclosed palatal wall of incisor canal. Mesial sides of central incisor teeth were seen. Flap sutured. Recovery uneventful. No complaints since.

Case VI

This case was first cited in my article, "Unusual Cysts of the Maxilla." As the follow-up history is most interesting, I cite it again.

History. Dr. M. G., age 31; complained of strange feeling in region of upper lip and nostril; slight fullness and tenderness experienced when upper lip was raised. Palpation over area, and over apices of upper incisors, elicited tenderness. Nasal examination negative. Patient in good health. Mouth examination, Sept. 10, 1927; all incisors present and in good condition. No evidence of dental work. Teeth normal in color; responded to heat and cold, and to electric pulp test. No history of trauma from patient or parents, both of whom are living. Radiograph revealed large area of decalcification in median line, with apices of teeth (incisors, central and lateral) apparently within this area of decalcification; had appearance of cyst, and lay between floor of nose and alveolar process. There appeared to be two cysts; larger one on right side (*fig. 16*).

Operation. Operative procedure was projected through mouth, although probably nerves of some teeth would be destroyed. After mucous membrane had been elevated on right side, high above apices of teeth, cyst membrane was seen directly underneath, through eroded buccal plate of bone. Cyst membrane then removed with difficulty. Membrane seemed to be held in mid-line. Throughout operation, patient complained of pain in naso-palatine region, especially when membrane was pulled forward. After operation probe could be passed through hole in median line of maxilla, from right to left side. Cyst contained large quantity of thick, brownish red, inspissated material. (Descending palatine artery passes through naso-palatine canal.) As it seemed we were dealing with two cysts, and as patient would not allow both sides to be opened at same time, that of right side was operated upon. Operative findings showed cyst to lie posteriorly to teeth and without connection with them, for at no time were apices of right incisor teeth seen. Several weeks later left side was operated upon; but after breaking through buccal plate high above apices of teeth, no cyst could be found. Evidently there was single cyst, and not double one as the radiograph seemed to indicate.

Follow-up. Immediately after operation patient had root canals of right central and lateral filled. On Dec. 11, 1929, patient came to office with following history: For past few days there had developed fullness in right ali nasi area. This morning he rubbed and pressed that side of nose, whereupon a profuse hemorrhage from nose ensued, lasting two hours. Just before patient came to office, had second hemorrhage. When he arrived, bleeding was from nasal orifice leading into incisor canal. Radio-

FIG. 13

FIG. 14

FIG. 13. Radiogram of Case IV, showing dentigerous cyst and incisor foramen.

FIG. 14. Photograph of cyst membrane and adherent soft-tissue content of incisor canal; Case IV.

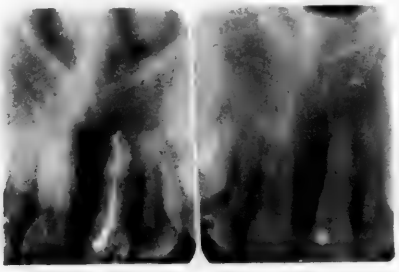


FIG. 15. Radiograms of Case V at first examination, and after root-canal filling but before operation.

FIG. 16

FIG. 17

FIG. 18

FIG. 16. Radiogram of Case VI.

FIG. 17. Radiogram of Case VI, two years later than fig. 16.

FIG. 18. Radiogram of Case VI, two and one-half years later than fig. 16.

graph of this region (*fig. 17*) showed that bone cavity formerly occupied by cyst had been filled in by new bone with exception of small area in center on right side. Following second hemorrhage, there have been no complaints. Radiograph taken July, 1930, shows further repair of bone in that area. (*fig. 18*).

Cases VII and VIII

Histology of incisor canal contents. Recently, two patients presented at Mt. Sinai Dental Clinic complaining of vague pains, in papilla palatine region, radiating upward into jaw and nose. One (Case VII) was Mr. H. F., age 45; other (Case VIII) was Mrs. R. H., age 35. Both were edentulous. Examination of each mouth disclosed alveolar ridge much re-absorbed, and papilla palatina very prominent, and projecting forward and upward. No abnormalities detected in radiographs of incisor region. Incisor foramen apparently normal. Since papilla would be subject to constant irritation by any denture, decided to remove contents of incisor canal, especially to study them microscopically. Tissue of canals easily removed without any hemorrhage, pain, or untoward complications.

Microscopic examination. Case VII.—Cross section shows tissue consists of fibrous connective tissue, in center of which are several blood vessels. Blood vessel walls thickened; nests of epithelial cells also visible. At one end of tissue, mucous glands present. Section not stained for nerve tissue. (*figs. 19* and *20*). *Case VIII.*—Cross section shows tissue consists of fibrous matrix with several blood vessels having very thick walls; nests of epithelial cells present. At one side, attached to it is portion of hard palate tissue with squamous epithelium. This section also not stained for nerve tissue (*figs. 21* and *22*).

VII. COMMENT

Case I exemplifies the simpler type of clinical pathology of this region, with the papilla palatina involved on two different occasions. After the simple treatment of puncturing the mass, drainage was re-established, and the results were favorable. No radiographic evidence of any changes in the incisor foramen could be demonstrated. Cases II, III, V, and VI represent the more complex types.

Case II presented with a definite cyst having very evident clinical signs of a tumor on the palate, and a history of a recurrence. The radiographic and operative findings agreed with the clinical signs. The microscopic evidence of a squamous epithelium that lined the

cyst cavity points to a persistent naso-palatine duct as the origin of this cyst. The father's history of a similar occurrence seems to corroborate its congenital nature.

Case III differs from Cases II and VI in that it originated, in my opinion, from the epithelial-cell rests found in this area. My belief is founded on the microscopic picture of the cyst membrane. Careful

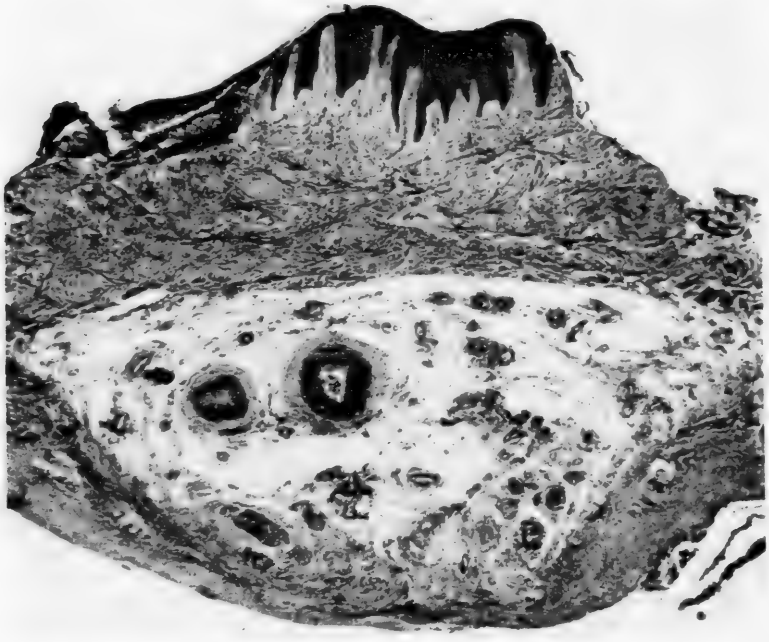


FIG. 19. Photomicrograph of soft tissue of incisor canal. Cross section; low power: Case VII. See fig. 20.

study of numerous sections of this tissue failed to show the presence of any squamous epithelium lining, but in the wall of the cyst are epithelial-cell rests, and mucous glands in groups are seen. Rushton (7), reported a similar case in a middle aged woman, edentulous, who complained of discomfort in the incisor region of the maxilla when wearing her denture. No abnormality was noted on inspection of the palate, but palpation disclosed that the palatal bone in the premaxillary region

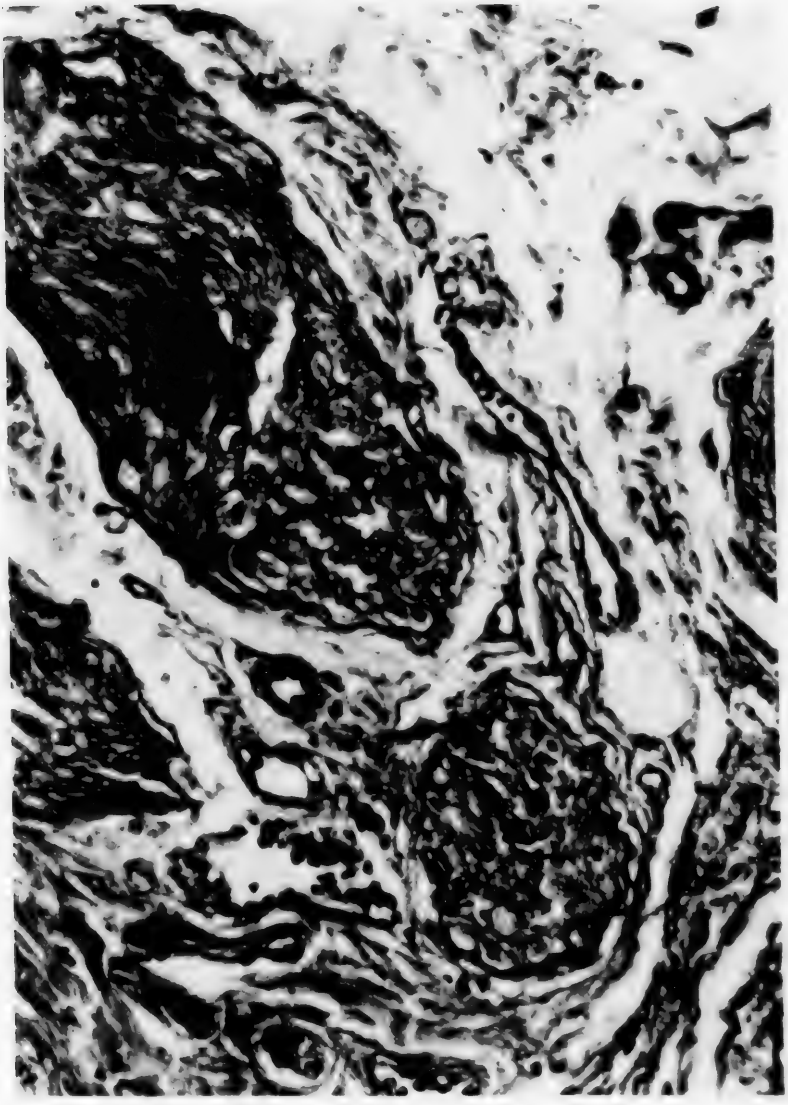


FIG. 20. Epithelial-cell rests; high power; Case VII. See fig. 19.

was absent. The radiographic findings agree in detail with that of Case III. Rushton, after ruling out the possibility that this cyst was due to either a simple root cyst or to the cystic degeneration of a germ of a supernumerary tooth, concluded that it probably was a sequestration (*fig. 23*). (Sequestration dermoids are one type of embryonic cyst that originate from misplaced epiblastic tissue and are found along the line of fusion in embryonic development, especially the facial and bronchial clefts.)

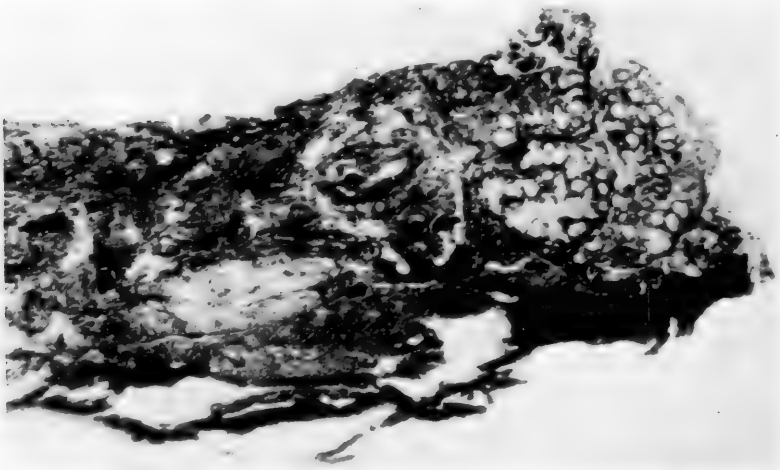


FIG. 21. Photomicrograph of incisor-canal contents; low power; Case VIII. See fig. 22.

Case IV demonstrates the presence of a dentigerous cyst whose growth sagitally caused the atrophy of the incisor canal wall, with subsequent adhesion to the soft tissue contents (*fig. 14*).

Case V represents a type of pathology of the incisor-canal contents that subsequently destroyed the anterior wall of the canal and produced a tumor under the buccal mucosa. The pyorrhea pocket along the mesial side of the central incisor raises the conjecture as to whether the paradentosis was primary and involved the incisor canal secondarily, or vice versa.

A history of an almost similar case, sent to me by Dr. H. A. Wild, of Basel (8), Switzerland, was reported in the "Around-the-table"

FIG. 22. Mucous glands; high power; Case VIII. See fig. 21.

talks in the *Dental Items of Interest:* "The patient, female, age 20, had a moderate swelling with a sinus over the spina nasalis anterior and root ends of the upper central incisors. She complained of neuralgic pains, radiating over the maxillae, along the sides of the nose, up to the eye and face. The operative findings were a granuloma at the apex of the right central incisor and a cyst in the incisor canal communicating" (*fig. 24*).

Case VI was first advanced by me as one originating from a naso-palatine duct. The subsequent interesting follow-up history, i.e., the hemorrhage escaping through the nasal orifice of the incisor canal, tends to confirm this conclusion.

FIG. 23

FIG. 24

FIG. 23. Radiogram demonstrating another case similar to Cases II and III, and to that described by Rushton.

FIG. 24. Radiogram of Dr. Wild's case, showing cyst of incisor canal and granuloma at apex of central incisor tooth.

In the cases of the cysts cited above, two pointed buccally (Cases V and VI); two (Cases II and III) pointed palatally.

VIII. DISCUSSIONS AND CONCLUSIONS

In my previous article, I reviewed the embryology of this region and quoted from Rawengel's histological studies on the relationship of the naso-palatine duct in the new born and the adult, in which he showed that persistent continuous or blind sacs from either the nasal or the mouth side were found frequently as anomalies. Piersoll, Sicher-Tandler, Leboucq, and Merkel (1) also mention the same condition. In addition to these persistent naso-palatine ducts or rem-

nants, Rawengal's studies further showed that the epithelial cell rests, derived from the middle epithelial cord, were found in the common incisor canal and in the posterior portion of the papillae.

Pathologic reactions within the soft-tissue contents of the incisor canal, whether produced by way of the papilla palatina or nasal route, can give rise to cyst formations in that area. This may be caused by (*a*) the occlusion of the lumen of a persistent naso-palatine duct or blind sac; or (*b*) by the pathologic reaction activating the growth of epithelial-cell rests contained in the soft tissue of the incisor canal, with their subsequent cystic degeneration; or (*c*) by the occlusion of the lumen of any mucous glands that may be found within the soft tissue of the incisor canal.

IX. SUMMARY OF CONCLUSIONS

The following practical conclusions can be drawn from these observations.

(1) The so-called "enlarged incisor foramina" may be pathologic conditions.

(2) "Enlarged incisor foramina," in the absence of symptoms, indicate that drainage of secretions is effectual.

(3) With a history of repeated swellings in the papilla palatina area, or of neuralgic pains radiating upward along the side of the nose and eye, operative interference is indicated. Conservative treatment by probing into the area, or by the use of drugs, gives temporary relief. Such conservative measures may be tried once. Radiograms should be used in checking up the case.

(4) The operative procedure is fairly simple and can be performed without fear of pain, hemorrhage, or untoward complications. (Pain is easily and effectually controlled by topical applications of a mixture of equal parts of 20 per cent cocaine hydrochloride and adrenalin solution.)

(5) In edentulous cases accompanied by symptoms of pain in the papilla region, since the usual "reliefs" in dentures for the papilla palatina are ineffectual, the removal of incisor-canal contents with its nerves produces favorable results.

(6) These cysts, occurring symmetrically in the median line and occupying the exact site of the incisor canal or foramen, are not of

dental origin. They may, owing to their proximity to the teeth, influence pathologic conditions about the teeth; or in turn, may be influenced by pathologic conditions of the teeth.

X. REFERENCES TO LITERATURE

(1) SCHROFF, JOSEPH 1929 Unusual cysts of the maxilla. *Dental Items of Interest*, li, p. 107.
(2) BLUM, THEODOR 1929 Cysts in the jaw. *Journal of the American Dental Association*, xvi, p. 649.
(3) PIERSOL. 1916 Human anatomy. 5th ed., ii, p. 1413; J. B. Lippincott, Philadelphia.
(4) SICHER-TANDLER. 1928 Anatomie für Zahnartz, p. 81; Julius Springer, Vienna.
(5) EULER, H. 1928 Die Entzündung der Papilla incisira (Superior) und der Canalis nasopalatinus. *Deutsche Zahnartzliche Wochenschrift*, xxxi, p. 686.
(6) HAUENSTEIN, K. 1929 Die entzündlichen Erkrankungen in Canalis nasopalatinus. *Ibid.*, xxxii, p. 372.
(7) RUSHTON, M. A. 1930 A cyst in the median palatine suture. *British Dental Journal*, li, p. 109.
(8) WILD, H. A. 1930 *Dental Items of Interest*, lii, p. 63.

267 West 81st Street

SUBPERIOSTEAL ABSCESSES ARISING FROM THE MANDIBULAR FIRST MOLAR[1]

HUGH W. MacMILLAN, D.D.S., M.D., F.A.C.D.

Cincinnati, Ohio

Of the many serious cases seen in oral surgery clinics and private practice the most frequent is the subperiosteal abscess. The term osteomyelitis has been loosely applied to most of these serious infections of the jaws. While it is true that the cortex of the bone and frequently the marrow cavity are involved secondarily, the general use of the term osteomyelitis confuses the rôle which the periosteum plays in confining the infection, in interfering with the nutrition of the bone, and in the extension of the abscess. General disturbances such as scarlet fever, diphtheria, and anemia may produce osteomyelitis and subperiosteal abscesses in the maxilla and mandible, even though there are no pulpless teeth or other evidence of dental infection. However, the chief cause is infection associated with the treatment or extraction of pulpless teeth, and the chief offending tooth is the mandibular first molar. The seriousness of some of these cases in children is graphically illustrated in the hospital records by the deaths reported as septicemia of dental origin. Meningitis caused by infections in the maxillary molar region occurs too often in medical literature. Since it is in the management of these cases that dentistry comes closest to surgery in a life and death struggle for the patient, it seems that a careful study of the conditions underlying this disease should be made, placing special emphasis upon prevention. In order to limit further the field of the discussion, only subperiosteal abscesses of the mandible will be considered.

The periosteum is the fibrous membrane that surrounds bones, and separates them from the environing tissue; it is nothing more. Where muscle fibers are inserted on bone, its fibrous organization is very different from that where bone is situated beneath mucous membrane

[1] Read before the Section on Oral Surgery at the Greater New York December meeting for Better Dentistry, New York City, December 12, 1929.

or the skin. In some regions there is absolutely no periosteum, muscle fibers inserting themselves directly on the bone; for instance, the linea aspera of the femur (1). The periosteum is like a closely adapted sac or elastic glove, clothing the surface of the bone, as if surrounding it in a fibrous bag, sewed firmly down at the margin of the cartilage around the articular ends. Besides the attachment around the cartilage, the periosteum is adherent where muscles or fascia are attached to the outer layer of the periosteum; where it approaches the insertion of tendons or ligaments; and where skin or mucous membrane seems attached to the underlying bone, as around the auditory meatus, the gums, mucous membrane of the nose, etc. In all such positions the periosteum is firmly attached to the bone—in fact becomes a part of it—and through this medium the connections between muscles, fascia, etc., and the framework of the skeleton, are accomplished (2). In adult life the periosteum is closely adherent to the shaft of the long bones. In children and adolescents while it is firmly attached to the extremities of the diaphyses, at the junction of the epiphyseal plates of cartilage; yet all over the shafts of the long bones, between these two plates, the periosteum is loosely adherent, and may be easily shelled from the bone. So loosely is it attached that in many instances a potential space may be described as existing between the inner layer of the periosteum and the bone. This potential space and its contained areolar tissue may be the sphere of physiological and pathological processes. When a pyogenic process has invaded the suoperiosteal areolar tissue, it spreads with great rapidity, converting the potential space into a pus-filled cavity with bare bone on one side and periosteum on the other, while at either extremity it is limited by the epiphyseal plates (3).

The surgical significance of the periosteum of the mandible may be recognized by a careful study of the muscle attachments that firmly bind the periosteum to the bone at these points. Between these muscle attachments the remainder of the periosteum is loosely adherent to the bone. In a virulent pyogenic process, pus may rapidly spread over the external surface of the mandible, being limited behind by the attachment of the masseter muscle, above by the buccinator muscle, anteriorly by the depressor muscles of the lip and the platysma. A review of the muscles of the body of the mandible will disclose that

along the lower border, between the attachments of the masseter muscle and the platysma, there is a broad open space to which nothing is attached except the thin layer of the superficial cervical fascia. Through this unguarded opening the subperiosteal abscess passes under the inferior border of the mandible, to invade the area under the loosely attached periosteum of the lingual surface; to be checked again in turn by the mylohyoid muscle (*fig. 3*). Owing to the number of muscle attachments of the anterior region of the mandible, with the attendant binding down of the periosteum, extensive subperiosteal abscesses are rarely seen, as the abscess usually finds egress from the bone above the muscle attachments. Another element undoubtedly is the pain from the pressure of the pus in these regions of greater density, which causes the patient to seek relief with greater dispatch.

The significance of the buccinator muscle attachment cannot be overstated in connection with alveolar abscesses arising from the mandibular first molar in children and young adults. If the pus travels along the joint between tooth and alveolus, as in *fig. 2*, finding egress from the bone above the attachment of the buccinator, an uncomplicated gum boil results, which is easily diagnosed and treated. But if on the other hand, it liquefies the bone and gains egress below the attachment of the buccinator, the pus spreads under the loosely attached periosteum, being directed downward and backward to the unprotected part of the border of the mandible immediately in front of the masseter muscle. Through this space it burrows its way subperiosteally, and gains admittance to that large region on the internal surface of the mandible below the mylohyoid muscle attachment. This broad space is covered with loosely attached periosteum, which is easily ripped up by the advancing pus. The abscess now is something of the shape of an hour glass, the constriction of the glass corresponding to that part of the abscess which passes through that strategic gateway just anterior to the masseter muscle. While alveolar abscesses of the mandibular teeth usually discharge externally, it not infrequently happens that abscesses from cuspids and premolars burrow lingually under the loosely attached periosteum of the internal surface. Limited by the mylohyoid muscle below, the pus spreads anteriorly, posteriorly, and toward the median line, ballooning out the space between the muscle and the mucous membrane of the sublingual space.

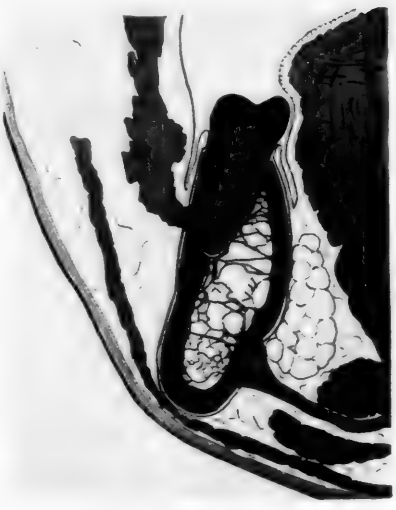


FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 1. CROSS SECTION IN THE REGION OF THE MANDIBULAR FIRST MOLAR

Buccinator and mylohyoid muscles are shown attached directly to the bone, thereby directing the extension of alveolar abscesses. See *figs. 2* and *3*.

FIG. 2. ENLARGEMENT OF FIG. 1

If the pus gains egress from the bone above the attachment of the buccinator muscle, an uncomplicated "gum boil" results, which is easily diagnosed. See *figs. 1* and *3*.

FIG. 3. SUBPERIOSTEAL ABSCESS IN REGION OF MANDIBULAR FIRST MOLAR

In children and adolescents a potential space exists between the periosteum and the bone. If pus gains egress below the attachment of the buccinator muscle, the abscess burrows downward and backward, raises the unattached periosteum just anterior to the masseter, passes to the internal surface and, if allowed to continue, is finally limited by the mylohyoid. See *figs. 1* and *2*.

FIG. 4. THE PREVENTIVE TREATMENT

The lancet is directed to a point lower than the root apices, and carried along the bone, thereby incising the periosteal envelope.

In the mandibular third molar region it is a point of great diagnostic value to recall the relation of the apex of the third molar to the lingual plate. Very frequently the apex extends through the bone into the

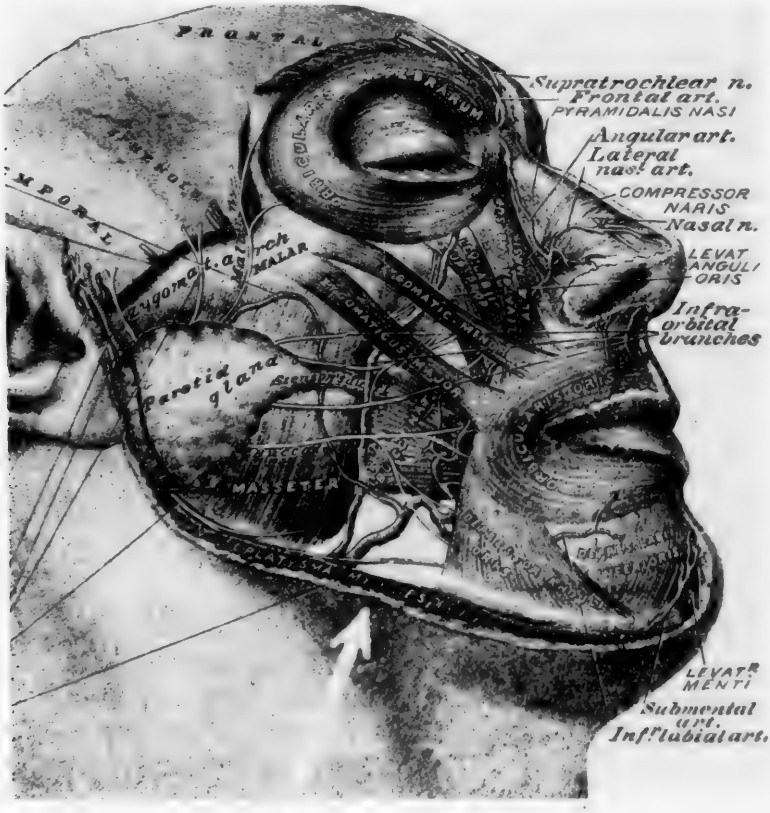


FIG. 5. THE STRATEGIC GATEWAY

The point of election for external drainage. (Illustration adapted from Practical Human Anatomy; Weisse.)

space below the mylohyoid ridge. A lingual subperiosteal abscess as a post-operative complication calls for discriminating knowledge of the anatomy of these parts. McEwen's description of the periosteum in children, "so loosely is it attached that in many instances a potential

space may be described as existing between the inner layer of the periosteum and the bone," is graphically illustrated in the following brief recital of a case history of a child eleven years of age. The influence of the buccinator and mylohyoid muscles in directing the extension of the abscess is also apparent.

October 6, 1927, L. B., aet. 11, complained of toothache in lower left first molar, which had been filled several weeks previously. Consulted family dentist when face began to swell, who advised waiting until swelling subsided. Contrary to dentist's expectations, swelling and pain increased. Family physician lanced and drained abscess externally. Offending molar finally extracted during following week. Five days following extraction, extensive swelling appeared on opposite side. No filled teeth on this side; no carious cavities; no history of dental disturbance. After few days of hot applications, external drainage again resorted to. Boy now had two external incisions, each draining pus and continued to drain for three months. During this time lateral jaw plates reported negative. Later when seen in consultation, careful examination of same plates revealed beginning involucrum, with no radiographic evidence of osteitis or osteomyelitis of body of bone. Case was clearly subperiosteal abscess. Examination with probe connected up large subperiosteal cavity along lower border of left jaw extending to mylohyoid muscle lingually. On right side identical condition was found. Both sides were connected by communicating channel located along lower border of mandible in region of symphysis.

The preventive treatment of these cases is obviously early drainage. No matter what the prejudice may be against extracting during the time the face is swollen, there is no contraindication for the establishment of drainage when pus has broken through the bone and created the subperiosteal abscess. Immediately an incision should be made in the buccal sulcus, in the region of the middle third of the mandibular first molar, and carried down in contact with the bone to a point lower than the apices of the tooth, or until pus is reached (*fig. 4*). The length of the incision depends upon the seriousness of the swelling. The use of gauze or rubber tubing for the maintenance of drainage depends upon the individual case. If drainage is secured, the extraction of the tooth may be delayed with impunity until the acute symptoms have disappeared. If the decision is made to extract the tooth at once, the extraction does not lessen the responsibility for establishing

external drainage of the subperiosteal infection. Any extension of the infection from the alveolus to the subperiosteal region calls for external drainage through the gum (4).

In far advanced cases of alveolar abscess of the mandibular first molar, with extensive swelling and fluctuation of the tissues of the submaxillary triangle, it becomes necessary to employ drainage extra-orally. From the foregoing description it will be recalled that the point of election for locating the pus is the region of the border of the mandible just anterior to the masseter muscle. A long incision from the median line to the ear, such as is practised by many general surgeons, is unnecessary and is not evidence of good surgical judgment. The attachments of the masseter, platysma myoides, depressor anguli oris, depressor labii inferioris, and levator menti, direct the pus to this point, which can easily be reached through a button-hole incision admitting the exploring hemostat. Rubber tubing to keep the opening patent, instead of gauze to plug it tightly, constitutes the final drainage measures.

CONCLUSIONS

1. The surgical anatomy of the buccinator and mylohyoid muscles must be appreciated in the rational treatment of complications arising from alveolar abscesses of the mandibular first molar.

2. The chief surgical significance of the unattached periosteum in the child is the necessity for prompt preventive treatment.

3. No matter what the prejudice may be against extracting during the time the face is swollen, there is no contraindication for the establishment of drainage when pus has broken through the bone and created the subperiosteal abscess.

REFERENCES TO LITERATURE

(1) LERICHE, R AND POLICARD, A. 1928 The normal and pathological physiology of bone, p. 107; C. V. Mosby Co., St. Louis.

(2) NOYES, F B. 1921 A text book on dental histology and embryology, p. 223; Lea and Febiger, Philadelphia.

(3) MCEWEN, WILLIAM 1912 The growth of bone, p. 24; James Maclehose and Sons, Glasgow.

(4) MACMILLAN, HUGH W. 1930 Diagnosis and treatment of post-extraction complications, *Journal of the American Dental Association*, xvii, p. 70.

1004 Union Central Building

WILLIAM J. GIES FELLOWSHIP IN BIOLOGICAL CHEMISTRY AT COLUMBIA UNIVERSITY[1]

EDGAR G. MILLER, JR., PH.D., *Chairman of the Fellowship Committee*

Department of Biological Chemistry, College of Physicians and Surgeons, Columbia University, New York City

Researches and publications

1. The research by the First Fellow (1928), Dr. James M. Inouye, entitled "Biochemical studies of salivary mucin," was published in the issue of this *Journal* for February 1930.[2]

2. Dr. Theodor Rosebury served as Second Fellow from July 1, 1928 to January 31, 1930, when he resigned to accept appointment as Instructor in Bacteriology in the College of Physicians and Surgeons, to continue and to extend, in the Department of Bacteriology, the research he had been doing as Second Gies Fellow. Dr. Rosebury's further research will be conducted on a grant by the Commonwealth Fund, to Columbia University, for a three-year study of the causes of decay of teeth. Dr. Rosebury's publications on research during his incumbency of the Fellowship are listed below:

1929 A simplified procedure for the separation and identification of protein in dental enamel (with Wm. J. Gies). *Journal of Dental Research*, ix, p. 299.

1929 A preliminary report on the relation of dental aciduric bacteria to *Lactobacillus acidophilus* (with R. W. Linton and L. Buchbinder). *Ibid.*, ix, p. 529.

1929 A comparative study of dental aciduric organisms and *Lactobacillus acidophilus* (with R. W. Linton and L. Buchbinder). *Journal of Bacteriology*, xviii, p. 395.

1930 A biochemical study of the protein in dental enamel. *Journal of Dental Research*, x, p. 187.

1930 Demonstration of methods for the separation and identification of proteins in enamel. *Ibid.*, x, p. 268.

[1] Previous reports relating to the Gies Fellowship were published in the *Journal of Dental Research:* (1) 1927, vii, p. 530; (2) 1928, viii, p. 679; (3) 1929, ix, p. 741.

[2] Inouye: *Ibid.*, 1930, x, p. 7.

1930 Studies of the aciduric bacteria of the mouth in relation to susceptibility and immunity to dental caries. *Journal of Dental Research*, x, p. 403.

3. Dr. Victor Ross, formerly an assistant in the Department, received the appointment as Third Fellow, to succeed Dr. Rosebury on February 1, 1930, and continues in service. He is conducting an inquiry into the fate of orally administered specific polysaccharide of pneumococcus. Among Dr. Ross' numerous researches was one published several years ago, conducted in part in this Laboratory, on "agglutinins in serum and saliva of rabbits inoculated with *B. acidophilus*."[3]

Register of Fellows

I. 1928 (Feb. 1–June 30): *James M. Inouye, B. S.* (Emory, 1915), M. A. (Columbia, 1917), Ph.D. (Columbia, 1925).

II. 1928–1930 (July 1–Jan. 31): *Theodor Rosebury*, D.D.S. (Pennsylvania, 1928).

III. 1930–1931 (Feb. 1– ?): *Victor Ross*, B. S. (College of the City of New York, 1916), M. A. (Columbia, 1918), Ph.D. (Columbia, 1923).

Financial statement

The report in 1928 contained the following comment (pp. 679–80): "It is a great pleasure to announce that Dr. Louise C. Ball, a candidate for the Ph.D. degree in the Department of Biological Chemistry in 1914 and 1915, has given to the Central Union Trust Company of New York City, in trust for the Fund, the sum of $10,000, the income therefrom, at 5 percent beginning in November 1928, to be paid quarterly by the Bank to Columbia University and added to the amount awarded directly to the then incumbent Fellows. *Dr. Ball reserves the right, in the event of personal disability or other personal emergency, to suspend one or more of these payments*, but has provided that in the event of her death during their continuance, the income shall be paid in perpetuity to the incumbent Fellows. The income from this gift will be approximately $300 for the remainder of the present academic year and $500 annually thereafter." [Italic not in the original.] Contributions of $300 in 1928–29, and $250 in 1929 to December 31, were paid by Dr. Ball on this plan. Last January Dr. Ball regretfully informed us that an emergency included in the foregoing reservation, which had been

[3] Ross, Krasnow, and Samet: *Journal of Dental Research*, 1927, vii, p. 337.

published with her approval, would make it impossible to continue the quarterly contributions to the stipend of the Fellow. The Committee records its hearty appreciation of Dr. Ball's generosity in contributing a total of $550 to the income of the Fellow in 1928–29 and 1929–30. An anonymous donor contributed $250, the remainder of Dr. Ball's intended contribution for 1929–30, to prevent reduction in the amount of the Fellow's stipend as assured for that year.

General financial summary

Principal of the William J. Gies Fellowship Fund, held by Columbia University on June 30, 1929, as recorded in the last annual statement[4]	$30,387.06
Additions to the principal of the Fund in 1929-30:	
By William N. Berg (second contribution)	50.00
One-half of a pro-rata distribution to special funds by Columbia University	139.55
Contributions by anonymous donors	139.55
Unexpended portion of the income (50 percent) in 1929–30	760.15
Total amount of the principal on June 30, 1930	$31,476.31

Stipend paid to the Fellows in 1929–30

Interest on the Fund as of June 30, 1930: $1,520.29. Of this amount, one half (by the terms of the original gift[5]) was added to the principal sum on June 30, 1930; the other half, paid to the Fellow	$760.14
Two quarterly contributions by Dr. Louise C. Ball	250.00
Payment by an anonymous donor to equal the balance that Dr. Ball had planned to contribute	250.00
One-half of a pro-rata distribution to special funds by Columbia University	139.55
Payment by anonymous donors (to raise the total to $1500)	100.31
Total amount paid to the Fellows	$1,500.00

[4] Miller: *Journal of Dental Research*, 1930, x, p. 743.

[5] Goodridge, Howe, Dunning, and Waugh: *Journal of Dental Research*, 1927, vii, p. 530. From 1928–1937, 50 per cent of the annual income will be added to the principal amount of the Fund; from 1938–1947, 30 per cent; from 1948–1957, 10 per cent. Beginning in 1958 (January 1) the annual income will be wholly available for current use.

Tentative estimate of the stipend for the Fellow in 1930–31

Interest on the Fund as of June 30, 1931: $1,573.82.[6] Of this amount, one-half (by the terms of the original gift[5]) will be added to the principal sum on June 30, 1931; the other half, paid to the Fellow.....	$786.91
Payments by anonymous donors (to date).....	125.00
Total amount payable to the Fellow.....	$911.91[6]

Additions to the principal of the Fund, or to the stipend of the Fellow, should be made payable to the Treasurer of Columbia University, and may be sent to the Treasurer of the University, to the Fellowship Committee, or to any officer of the Biochemical Department of Columbia University.

October 31, 1930

[6] Prospective additional contributions during the year may increase this amount.

SUPPLEMENT

CONTENTS

I. New York Academy of Dentistry. Notes from the minutes of the proceedings in 1929–30. *Malcolm W. Carr*, D.D.S., *Editor* 775
II. William Jarvie Society for Dental Research. Notes from the minutes of the proceedings in 1929–30. *Joseph Schroff*, M.D., D.D.S., *Editor* 781
III. International Association for Dental Research. Memoranda on the proceedings in 1929–30. *William J. Gies*, F.A.C.D., *Editor* 782
IV. Important books recently received by the *Journal of Dental Research* 786

I. New York Academy of Dentistry

NOTES FROM THE MINUTES OF THE PROCEEDINGS IN 1929–30

Malcolm W. Carr, D.D.S., Editor

OFFICERS

President, S. E. Davenport, Jr.; *Vice-president*, Leuman M. Waugh; *Secretary*, Lowrie J. Porter; *Treasurer*, Alfred L. Kohn; *Editor*, Malcolm W. Carr.

Executive Committee: Albert J. Irving, Edwin G. Van Valey, George M. Babbitt, *Chairman.*

Fellowship Committee: William B. Dunning, Harold S. Vaughan, Sebert E. Davenport, *Chairman.*

Non-resident Fellowship Committee: Sebert E. Davenport, Harold S. Vaughan, William B. Dunning, *Chairman.*

Dental Center Committee: William J. Gies, Arthur H. Merritt, Bissell B. Palmer, *Chairman.*

Research Council: Charles F. Bödecker, Henry W. Gillett, Arthur H. Merritt, Leuman M. Waugh, Bissell B. Palmer, *Secretary*, William J. Gies, *Chairman.*

Activities Bureau: Edwin J. Nestler, Eugene Senior, Edwin G. Van Valey, George S. Callaway, *Chairman.*

Advisory Committee of the Activities Bureau: Charles F. Bödecker, William J. Gies, Arthur H. Merritt, Leuman M. Waugh, Bissell B. Palmer, *Chairman.*

SCIENTIFIC MEETINGS

During the season of 1929–30, the Academy held six regular meetings, and a special one, at the Yale Club, New York City, and one special meeting at the New York Academy of Medicine. All except the latter were preceded by informal dinners at the Yale Club.

October. George H. Semken, M.D., New York City, presented a paper on "tumors of the upper jaw, with special reference to cancer." Dr. Harold S. Vaughan discussed the paper from the standpoint of the oral surgeon; Dr. Edward Kennedy, from the standpoint of prosthodontia.

November. Two papers were read in a symposium on prosthetic dentistry.[1] Norman Essig, D.D.S., Philadelphia, Pa., considered "esthetics in denture construction," which was discussed by Dr. Russell W. Tench. Irving R. Hardy, D.M.D., New York City, presented "pressure balance in denture construction," which was discussed by Dr. Martin L. Collins.

January. In a symposium on "the relation between medicine and dentistry,"[2] papers were presented by Russell Cecil, M.D., New York City, and Leroy M. S. Miner, D.M.D., M.D., Boston, Mass. Dr. Cecil spoke from his experience as a physician; Dr. Miner, from the dental viewpoint. No formal discussion had been pre-arranged, but, as had been hoped, a free discussion ensued in which both physicians and dentists participated.

February. Leuman M. Waugh, D.D.S., New York City, delivered an address on "the nutrition of the Eskimo of the North Bering Sea and Arctic Alaska, with special reference to the teeth."[3]

March. The fifth annual series of progressive table clinics was presented as follows: (*a*) "Esthetics in dentures," by Dr. Irving R. Hardy. (*b*) "The spring-lock insertion bridge; pictures and made-up cases demonstrating an innovation of the removable bridge, which, though it eliminates resistance to removal and insertion by the patient, cannot possibly become displaced during mastication," by Dr. Ferdinand G. Neurohr. (*c*) "Essential considerations for the

[1] Essig, Norman, and Hardy, Irving R.: *Journal of Dental Research*, 1930, x, p. 569.

[2] Cecil, Russell, and Miner, Leroy M.S.: *Journal of Dental Research*, 1930, x, p. 137.

[3] An abstract of this work, as reported to the International Association for Dental Research, has been published: *Journal of Dental Research*, 1930, x, p. 387.

general practitioner in determining the correct time for orthodontic treatment in various types of malocclusion," by Dr. Lowrie J. Porter. (*d*) "Removable-bridge work; a completed case, with progressive models demonstrating the principles involved," by Dr. Edwin G. Van Valey.

April (*special meeting*). On April 3, 1930, at the regular open meeting of the New York Academy of Medicine, the New York Academy of Dentistry met jointly with the Section on Ophthalmology of the Academy of Medicine, a symposium being presented on "oral infections and their relation to diseases of the eye."[4] "The viewpoint of the ophthalmologist" was presented by Dr. W. F. C. Steinbugler, New York City; "the viewpoint of the general practitioner of dentistry," by Dr. Henry W. Gillett, New York City; "the viewpoint of the oral surgeon," by Dr. Henry S. Dunning, New York City. There were discussions of the medical aspects of the subject by Drs. Conrad Berens and James G. Dwyer; of the dental aspects, by Drs. Arthur H. Merritt and John Oppie McCall.

April (*annual meeting*). Reports of officers and committees were presented. The following officers were re-elected for the coming year: *President*, S. E. Davenport, Jr.; *Vice-president*, Leuman M. Waugh; *Secretary*, Lowrie J. Porter; *Treasurer*, Alfred L. Kohn; *Editor*, Malcolm W. Carr.

Two associate fellows, Dr. George S. Callaway and Dr. Earl Banks Hoyt, were elevated to active fellowship in the Academy, in recognition of achievement and distinction in professional activities.

A report of a new study by the Research Council, on five so-called tooth-cleaning fluids, was presented by Drs. William J. Gies, Charles F. Bödecker, and Maxwell Karshan.[5]

Dr. Arthur Frank Payne addressed the Academy on "psychology applied to dentists and the practice of dentistry." There was an open discussion following Dr. Payne's address.

June (*special meeting*). Harold F. Hawkins, D.D.S., Los Angeles, Calif., presented a paper based upon his researches, entitled, "A

[4] Steinbugler, W. F. C., Gillett, Henry W., and Dunning, Henry S.: *Journal of Dental Research*, 1930, x, p. 685.

[5] Research Council of the New York Academy of Dentistry: *Journal of Dental Research*, 1930, x, p. 481.

rational technic for the control of caries and systemic pyorrhea."[6] Dr. Charles F. Bödecker, New York City, presented a paper on "a new theory of the cause of dental caries." Discussion was opened by Dr. William J. Gies and continued by Drs. Bödecker and Hawkins.

COMMENT

During the past year the Academy engaged in a number of activities that are worthy of special mention. Two meetings, devoted to medico-dental discussions, were held to foster closer coöperation between medicine and dentistry. These were well attended by representative groups of physicians and dentists.

The Academy initiated the custom of receiving, at each meeting, a short informal presentation of "incidents of office practice." Fellows volunteer to present incidents of practice with which they have become familiar, and which may be of interest to the group. The presentations are usually practical in their application. This feature has aroused much interest and has many advantages.

The Activities Bureau, in addition to its usual work of placing fellows in contact with professional activities in which they may wish to participate, has extended the scope of its work to include the preparation of a catalog, which will record the past professional relationships of the entire fellowship. Additional data will be annually included in these records, which thus will present complete information on each fellow's activities, interests, and accomplishments.

The report of the Research Council was presented at the annual meeting by the *Chairman*, Dr. William J. Gies, who said, in part:

"The Research Council was organized at the April meeting in 1927. It was established to conduct researches to be suggested by the members of the Council and the Academy It began its work by emphasizing the need for studies of various claims advertised for dental industrial products. Reports were published in 1927 and 1929;[7] another is about to be published.[6] You noted from your Treasurer's report that $3,454 remained in the treasury to the credit of the Research Council, and that only a very small sum was used during the past year. An outstanding feature in the work of the Council is the generosity that has been shown at the Dental and Medical

[6] To be published in the issue of the *Journal of Dental Research* for February, 1931.

[7] Research Council of the New York Academy of Dentistry: *Journal of Dental Research*, 1927, vii, p. 477; *Ibid.*, 1929, ix, p. 171.

[9]Schools of Columbia University in support of the research under the auspices of this body. Not only at the older sites, but also at the new, the officers of the Department of Oral Histology and Embryology in the Dental School, and of the Department of Biological Chemistry in the Medical School, have given the various necessary supplies without expense to the Council. Drs. Bödecker, Applebaum, and Karshan have performed the experimental work under conditions that have required very little of the Council's funds; in fact, if our colleagues had been paid honoraria in accord with their deserts, there would be no money to our credit in the treasury. Their generosity has made it possible to announce that the contributions you intended to pay to the Research Council this year will not be collected, unless the Council's program should be expanded into unexpected dimensions."

CORRECTION OF A PUBLIC MISSTATEMENT REGARDING THE PUBLICATION OF THE PROCEEDINGS IN THIS JOURNAL

The *Journal of the American Dental Association* published in the number for September, 1930 (pages 1744–1752), a report of an investigation by the Council on Dental Therapeutics on "the Sherman L. Davis Treatment." This report, which includes an investigation of Dr. Davis' contributions to dental periodical literature, states that "the *Journal of Dental Research* carries a report of an address delivered by Dr. Davis before the New York Academy of Dentistry, in January 1928." To this assertion there is appended a footnote as follows: "The dean of the Indiana University School of Dentistry, in a communication which was referred to the Council, denies that Dr. Davis contributed an article to the *Journal of Dental Research*. He stated that the address was printed from stenographic notes." This statement by the Dean appears to rest wholly on assurances given him by Dr. Davis.

The essential facts are these: A transcipt of the stenographic notes of Dr. Davis' address before the Academy, on January 23, 1928, was forwarded to Dr. Davis with the request that he return the manuscript for publication after making the usual editorial revision and correction. Dr. Davis returned a corrected and retyped copy of this manuscript, which was published in the *Journal of Dental Research*.[8] The original manuscript, as revised by Dr. Davis, is now in the files of the Editor of the Academy.

[8] Davis, Sherman L.: The relation of nutrition to general immunity. *Journal of Dental Research*, 1929, ix, p. 11.

IN MEMORIAM

Bissell B. Palmer, Sr.

The following memorial and resolution were adopted at the January meeting:

"The death of Dr. Bissell Barbour Palmer, Sr., on July 5, 1929, terminated an interesting and active life. He was born on November 4, 1860, in the Canadian town of Sackville, Province of New Brunswick.

"Early in life, and following his school days in Dorchester, he secured employment with the C. P. Railroad, soon learning telegraphy, later becoming an expert cable operator at Halifax. Transferred to the New York office of the Western Union (cable department), he remained with them a number of years.

"During this period, taking advantage of every opportunity, he entered the New York College of Dentistry, graduating with the class of 1886, of which he was one of the few surviving members. He began the practice of dentistry in West 34th St., opposite the present site of Macy's, later removing to Broadway and 53rd Street.

"During these days he was an active worker in Republican city politics, extending his already large circle of friends. He was a life-long member of the First District Dental Society of the State of New York, always supporting its advancing policies, and upon the formation of the New York Academy of Dentistry, was elected as its first Associate Fellow.

"He was married to Margaret Doherty of St. Johns who survives him. Their only son, Bissell B. Palmer, Jr., is a charter member of this Academy.

"Dr. Palmer always stood for the best in dentistry, for higher standards in education, and for professional journalism. His was a personality that attracted friendships, and his faculty for choosing the brighter side of life, his whimsical philosophy, his inexhaustible fund of anecdotes and personal reminiscences, endeared him to all who knew him. His death was cause for sincere sorrow."

"Your Committee offers the following resolutions:"

"*Whereas*. God in his Wisdom has called our good friend and fellow Academician; therefore, be it

"*Resolved*, That this Academy does hereby express its grief at his loss, and extends the sincere sympathy of its members to his family; and, be it further

"*Resolved*, That this memorial and resolution be inscribed upon the records of this Academy and a copy be sent to the family."

(*Signed*) Leland Barrett

II. William Jarvie Society for Dental Research

NOTES FROM THE MINUTES OF THE PROCEEDINGS IN 1929–30[9]

Joseph Schroff, M.D., D.D.S., Editor

During 1929–30 the Society held five scientific meetings, and a number of business meetings, at the School of Dental and Oral Surgery of Columbia University.

SCIENTIFIC PROGRAMS

December 13, 1929. Dr. Leuman M. Waugh: A study of the teeth of the Eskimo of the North Bering Sea and Arctic Alaska.[3]

January 31, 1930. Dr. W. H. Crawford: The physical properties of dental materials.

February 28, 1930. Dr. Henry W. Gillett: The relations of inlay margins to cavity margins.

March 28, 1930. Dr. Henry S. Dunning: Some recollections of maxillo-facial surgery during the war.

May 2, 1930. Dr. Joseph Schroff: Further studies on the pathology of the incisive foramen and naso-palatine regions.[10]

EXECUTIVE PROCEEDINGS

At the annual business meeting, on May 2, 1930, an amendment of the Constitution was adopted to the effect that undergraduate membership shall be limited to all seniors who have shown interest in the work of the Society, and fifteen juniors, to be chosen on a basis of (*a*) scholarship, and (*b*) eligibility as useful members.

OFFICERS IN 1930–31

President: Nathan P. Berger. *Vice-president:* Joseph D. Kudler. *Secretary-treasurer:* Ben F. Daneman. *Editor:* Joseph Schroff.

[9] Recorded by James M. Dunning, Secretary, 1929–30.

[10] Schroff: *Journal of Dental Research*, 1930, x, p. 739.

III. International Association for Dental Research memoranda on the proceedings in 1929–30[11]

William J. Gies, F.A.C.D., Secretary

EIGHTH GENERAL MEETING

The eighth general meeting of the International Association for Dental Research was held in Toronto, Canada, at the Royal York Hotel, March 24–26, 1930, in coördination, by agreement, with the sessions of the seventh annual meeting of the American Association of Dental Schools. The members of each body were welcome at, and participated in, all sessions of both associations. In the absence of the President, Dr. Arthur D. Black, Dental School, Northwestern University, who was en route to Japan, the chairmen were the Vice-president, Dr. A. E. Webster, Dental School, University of Toronto, and the President-elect, Dr. U. G. Rickert, Dental School, University of Michigan. The Association conducted informal dinners in the Royal York Hotel for members and guests on the evenings of March 24 and 26. Forty-eight members and a larger number of visitors attended each of the four scientific sessions, where fifty-four papers were presented. Abstracts of these papers were published on pages 372–419 of this volume. The session on the evening of March 25 was devoted to the President's address, which, in Dr. Black's absence, was presented for him by Dr. E. H. Hatton,[12] and to executive matters. The colleagues listed on pages 421–424 of this volume were elected to membership. There are now 228 members and fifteen sections (page 786).

The meeting was not only the largest in attendance of members and guests, but also the most enjoyable in informal features, the most

[11] A brief outline of the organization and early development of the International Association for Dental Research, including the Constitution, was published on pages 197–208 of Volume VIII (1928) of the *Journal of Dental Research*. This was followed, in the same issue, by a summary of the proceedings of the general meetings from 1920 to 1927, inclusive, and a register of membership as of March 15, 1928 (pp. 209–233). Records of the proceedings were published in the *Journal of Dental Research* as follows: *Sixth* general meeting (1928)—Volume VIII—abstracts, page 395; executive proceedings, page 696. *Seventh* general meeting (1929)—Volume IX—abstracts, p. 269; executive proceedings, p. 756. *Eighth* general meeting (1930)—Volume X—abstracts, p. 367.

[12] Black: *Journal of Dental Research*, 1930, x, p. 367.

fruitful in scientific presentations, and the most instructive and useful in the history of the Association. The papers on the program covered a broad field of interest, and the experiments described therein included an unusual range of accurate technical procedures. The following statistical data indicate the quality of the program, the wide geographical distribution of the participants, and the spread of interest in dental research.

Academic and professional degrees of the participants.—Bachelor, 22. Master, 11. D.D.S. (D.M.D.), 40. M.D., 19. Ph.D., 5.

Universities from which papers were presented.—Columbia (7), Dalhousie (2), Harvard (1), Hawaii (3), Johns Hopkins (1), Loyola—Chicago (2), Michigan (2), New York (3), Northwestern (3), Pennsylvania (4), Pittsburgh (1), Toronto (2), Tufts (1), Vienna (8), Western Reserve (4), Yale (5).

Independent medical schools or hospitals from which papers were presented.—Fifth Avenue Hospital, New York (1), Jefferson Medical College (1), Mayo Clinic (1), Peiping Union Medical College (1), Philadelphia General Hospital (1).

Countries represented by the participants.—Austria (8), Canada (6), China (1), United States 39 (Hawaii, 4).

Sections represented by participants.—Ann Arbor (2), Boston (2), Chicago (5), Halifax (2), New York (13), Philadelphia (12), Pittsburgh (1), Toronto (4), Vienna (8).

SECTIONS

The sections in existence at the date of the general meeting in Toronto were those in Ann Arbor, Boston, Chicago, Halifax, Minnesota, New York, Philadelphia, Pittsburgh, San Francisco, St. Louis, Toronto, and Vienna. Since that meeting sections have been organized in Cleveland, Ohio; New Haven, Connecticut; and Winnipeg, Manitoba.

During 1929–30 meetings of the sections were held on this schedule: *Ann Arbor*, May 22. *Boston:* November 5, February 3, May 27. *Cleveland:* May 14. *Chicago:* December 16, February 26, May 8. *Halifax:* October 21, May 2. *New Haven:* March 29. *New York:* December 5, March 13. *Philadelphia:* October 30, November 13. *Pittsburgh:* November 4, December 2, January 6, February 3, March 3,

April 7, May 5, June 2. *San Francisco:* October 4. *St. Louis:* September 12, October 10, November 14, December 12, January 9, February 13, March 13, April 10, May 8, June 12. *Toronto:* October 2, January 7, February 20. *Vienna:* September 21. *Winnipeg:* April 23.

Proceedings of sectional meetings have been published on pages 1, 113, 133, 247, 265 and 443 of this volume.

NEXT GENERAL MEETING

The ninth general meeting will be held in Memphis, Tennessee, on March 21 and 22, preceding and in coördination there with the next annual meting of the American Association of Dental Schools on March 23–25, inclusive. The scientific sessions of the Association and of its sections are open to visitors.

REGISTER OF MEMBERS AND OFFICERS: DECEMBER 1, 1930

Members

1. *Ann Arbor Section.*—R. W. Bunting, Faith P. Hadley, Philip Jay, C. J. Lyons, **U. G. Rickert*, E. O. Scott, M. L. Ward—7

2. *Boston Section.*—L. W. Baker, **F. R. Blumenthal*, W. G. Bridge, F. P. Chillingworth, H. DeW. Cross, F. H. Daley, Adelbert Fernald, C. E. Hatch, E. A. Hooton, P. R. Howe, A. L. Johnson, V. H. Kazanjian, P. A. Leavitt, L. M. S. Miner, A. L. Morse, F. W. Morse, William Rice, A. P. Rogers, Benjamin Spector, K. H. Thoma, Benjamin Tishler, Harry Trimble, G. H. Wright—23

3. *Chicago Section.*—Lloyd Arnold, Willam Bebb, H. C. Benedict, Olaf Bergeim, A. D. Black, J. R. Blayney, E. P. Boulger, E. D. Coolidge, D. J. Davis, R. H. Fouser, T. L. Gilmer, E. H. Hatton, **Rudolf Kronfeld*, H. A. McGuigan, F. B. Moorehead, Emil Mueller, Anna C. Nichols, F. B. Noyes, V. T. Nylander, W. F. Peterson, H. A. Potts, B. O. Sippy, W. G. Skillen, S. D. Tylman, W. H. Welker—25

4. *Cleveland Section.*—**S. W. Chase*, T. J. Hill, V. C. Myers, F. C. Waite—4

5. *Halifax Section.*—J. S. Bagnall, R. J. Bean, W. G. Dawson, A. W. Faulkner, W. C. Oxner, S. G. Ritchie, R. P. Smith, **G. K. Thomson*, E. G. Young—9

* Secretary of the Section.

6. *Minnesota Section.*—E. S. Best, J. T. Cohen, I. A. Epstein, B. S. Gardner, C. J. Grove, H. A. Maves, J. F. McClendon, F. S. Meyer, G. S. Monson, W. D. Vehe, **C. W. Waldron*, H. G. Worman, D. E. Ziskin—13

7. *New Haven Section.*—C. G. Burn, A. M. Crosby, R. G. Hussey, **W. G. Downs, Jr.*, J. P. Pigott, G. H. Smith—6

8. *New York Section.*—B. G. Anderson, Edmund Applebaum, Adolph Berger, Theodor Blum, C. F. Bödecker, L. R. Cahn, M. W. Carr, H. E. S. Chayes, G. W. Clapp, H. S. Dunning, W. B. Dunning, J. D. Eby, S. W. A. Franken, W. J. Gies, H. W. Gillett, W. K. Gregory, Milo Hellman, Isador Hirschfeld, R. G. Hutchinson, Jr., T. P. Hyatt, J. M. Inouye, Maxwell Karshan, F. C. Kemple, Frances Krasnow, H. J. Leonard, J. O. McCall, F. S. McKay, A. H. Merritt, E. G. Miller, Jr., H. F. Osborn, **B. B. Palmer*, Theodor Rosebury, J. P. Ruyl, M. I. Schamberg, Joseph Schroff, P. R. Stillman, L. M. Waugh, B. W. Weinberger, J. L. Williams, J. L. Young—40

9. *Philadelphia Section.*—J. L. T. Appleton, Jr., **Mrs. C. K. Bryant*, T. J. Cook, J. A. Detlefsen, R. H. Ivy, Joseph McFarland, J. V. Mershon, W. R. Pentz, S. E. Pond, Hermann Prinz, A. Hopewell-Smith, C. R. Turner—12

10. *Pittsburgh Section.*—T. W. Brand, Kaethe W. Dewey, F. C. Friesell, H. E. Friesell, W. F. Swanson, **L. E. Van Kirk*, W. H. Wright—7

11. *St. Louis Section.*—W. A. Chamberlain, E. H. Keys, E. B. Owen, H. R. Raper (Albuquerque, N. M.), **C. O. Simpson*, R. C. Wheeler, J. D. White, G. B. Winter—8

12. *San Francisco Section.*—G. L. Bean, B. E. Lischer, J. A. Marshall, E. H. Mauk, G. S. Millberry, F. H. Orton, **J. S. Shell*, F. V. Simonton—8

13. *Toronto Section.*—J. F. Adams, G. R. Anderson, H. K. Box, **A. J. Broughton*, Thomas Cowling, W. E. Cummer, A. W. Ellis, C. Jane Fraser, G. W. Grieve, Andrew Hunter, F. C. Husband, A. J. McDonagh, E. W. Paul, R. J. Reade, H. S. Thomson, W. C. Trotter, A. E. Webster—17

14. *Vienna Section.*—Arved Berg, Karl Breitner, Fritz Driak, Bernhard Gottlieb, Richard Grohs, Otto Hofer, Ernst Kellner, **Emerich Kotanyi*, Moriz Leist, Balint Orban, Hans Pichler, Otto Preisecker, Franz Schönbauer, A. M. Schwarz, Harry Sicher, Karl Spring, George Stein, Richard Trauner, Josef Weinmann, Herman Wolf—20

15. *Winnipeg Section.*—**E. R. Bier*, F. T. Cadham, M. H. Garvin, K. M. Johnson, Anne G. E. Mackenzie, H. J. Merkeley, W. J. Riley—7

16. *Not members of a section: Baltimore.*—Henry Klein, E. V. McCollum. *Germany.*—Wilhelm Dieck, Friedrich Proell, Rudolf Weber. *Honolulu.*—Martha R. Jones, R. W. Leigh. *Indianapolis.*—S. L. Davis. *Iowa City.*—

* Secretary of the Section.

A. W. Bryan, C. L. Drain, R. H. Volland. *London.*—Arthur Bulleid, Frank Colyer, F. N. Doubleday, E. W. Fish, Evelyn Sprawson, J. G. Turner. *Louisville.*—T. B. Beust. *Washington.*—A. B. Crane, R. A. Keilty, F. E. Rodriguez, Wilmer Souder—22

Total number of members—228

OFFICERS: 1930–31

President—U. G. Rickert, University of Michigan.
President-elect—A. E. Webster, University of Toronto.
Vice-president—R. W. Bunting, University of Michigan.
Treasurer—William Rice, Tufts College.
Secretary—William J. Gies, Columbia University.

IV. Important Books Recently Received by the Journal of Dental Research[13]

Rickets including osteomalacia and tetany. By Alfred F. Hess, M.D., Clinical Professor of Pediatrics, University and Bellevue Hospital Medical College, New York City. 1929: pp. 485; 7¼ x 4 in.; 52 illustrations; $5.50. Lea and Febiger, Philadelphia, Pa.

Synopsis of the practice of preventive medicine as applied in the basic medical sciences and clinical instruction at the Harvard Medical School. By the Faculty of Medicine. 1929: pp. 194; 6¼ x 3⅝ in.; no illustrations. Harvard University Press, Cambridge, Mass.

La pratique stomatologique. IV. *Dentisterie opératoire.* By Pol Nespoulous, D.D.S. (College dentaire de Chicago), Professor à l'Ecole Francaise de Stomatologie; Stomatologiste des Hôpitaux de Paris. 1929: pp. 541; 6¾ x 4 in.; 263 illustrations; 65 fr. Masson et Cie, Paris, France.

Ueber die Natur der muskelrheumatischen (myalgischen) Schmerzsymptome. By Folke Lindstedt, M.D., Stockholm, Sweden. Supplement XXX of *Acta Medica Scandinavica*, lxxii. 1929: pp. 180; 7 x 4 in.; 13 illustrations.

Testicular grafting from ape to man. By Serge Voronoff, Director of the Laboratory of Experimental Surgery of the Collège de France, and Assistant Director of the Laboratory of General Biology of the Ecole des Hautes Études; and George Alexandrescu, formerly assistant at the Institut d'Histologie, and Staff Hospital Physician, Bucharest, Roumania. *Translated* by Theodore C. Merrill, M.D. 1929: pp. 125; 6¼ x 3⅞ in.; 39 illustrations. Brentano's Ltd., London, England.

[13] The sizes of the pages, exclusive of margins, are given in inches.

The properties and uses of drugs. By HENRY H. RUSBY, Ph.M., M.D., D.Sc., Dean, and Professor of Materia Medica, College of Pharmacy, Columbia University; A. RICHARD BLISS, Jr., A.M., Phar. D., M.D., Dean of the School of Pharmacy, and Chief of the Division of Pharmacology and Materia Medica, Colleges of Medicine and Dentistry and School of Pharmacy, University of Tennessee; and CHARLES W. BALLARD, A.M., Phar. D., Ph.D., Associate Professor of Materia Medica and Director of the Microscopical Laboratory, College of Pharmacy, Columbia University. 1930: pp. 823; 7¾ x 4⅝ in.; 38 illustrations; $6.50. P. Blakiston's Son and Co., Philadelphia, Pa.

Public health aspects of dental decay in children. Number III of School Health Research Monographs. By RAYMOND FRANZEN, Ph.D., Director of Research, School Health Study, American Child Health Association. 1930: pp. 121; 7 x 4½ in.; 2 charts, 10 graphs, and many tables; $2.00. American Child Health Association, New York.

Étude sur l'éclosion épidémique de l'influenza. By JOHS. MYGGE. Supplement XXXII of *Acta Medica Scandinavica*, lxxiii. 1930: pp. 145; 7 x 4 in.; 5 illustrations.

Zur Kenntniss der lokalisirten Thrombenbildungen in der Vena iliaca communis sinistra. By ANDERS KRISTENSON. Supplement XXXIII of *Acta Medica Scandinavica*, lxxiii. 1930: pp. 82; 7 x 4 in.; 28 illustrations.

An outline of oral surgery for the general dental practitioner. By JAMES L. ZEMSKY, D.D.S., Attending Oral Surgeon to the Midtown Hospital, New York. 1930: pp. 171; 6⅞ x 4 in.; 116 illustrations; $4.50. Physicians and Surgeons Book Co., Brooklyn, N. Y.

Oral diseases. A practical treatise offering diagnostic and therapeutic aid to the practitioners of medicine and dentistry. By JAMES L. ZEMSKY, D.D.S., Attending Oral Surgeon to the Midtown Hospital, New York. 1930: pp. 402; 6¾ x 4 in.; 414 illustrations; $8.00. Physicians and Surgeons Book Co., Brooklyn, N. Y.

Necrotic gingivitis (trench mouth). By HAROLD KEITH BOX, D.D.S., Ph.D., Research Professor of Periodontology, Faculty of Dentistry, University of Toronto. 1930: pp. 64; 7 x 4⅛ in.; 18 illustrations. University of Toronto Press, Toronto, Canada.

Medical education and related problems in Europe. By WILLARD C. RAPPLEYE, Director of the Study by the Commission on Medical Education. 1930: pp. 200; 7⅛ x 4 in.; no illustrations. Published by the Commission, 215 Whitney Ave., New Haven, Conn.

Those teeth of yours. By J. MENZIES CAMPBELL, L.D.S., D.D.S., F.R.S.E., F.I.C.D., Fellow of the Royal Society of Medicine. 1929: pp. 141;

5½ x 3¼ in.; 17 illustrations; 3/6 net. William Heinemann, Ltd., 99, Great Russell St., London, W. C. I, England.

Infant nutrition. By WILLIAM MCKIM MARRIOTT, B.S., M.D., Professor of Pediatrics, School of Medicine, Washington University. 1930: pp. 375; 7⅝ x 4⅝ in.; 55 illustrations; $5.50. C. V. Mosby Company, St. Louis, Mo.

The physiology of oral hygiene and recent research, with special reference to accessory food factors and the incidence of dental caries. By J. SIM WALLACE, D.Sc., M.D., L.D.S., Lecturer on Preventive Dentistry, King's College Hospital; formerly Dental Surgeon and Lecturer on Dental Surgery and Pathology, London Hospital. *Second edition.* 1929: pp. 228; 6¼ x 3½ in.; 7 illustrations. Baillière, Tindall and Cox, 7 and 8, Henrietta St., Covent Garden, London, England.

Christian and Jew. A symposium for better understanding. By thirty-six contributors; edited by ISAAC LANDMAN, 1929: pp. 374; 6⅛ x 3½ in.; 37 illustrations (portraits). Horace Liveright, New York.

Higher education in America. Edited by RAYMOND A. KENT, President of the University of Louisville, with twenty-two collaborators. (Chapter V: *The school of dentistry*, by WILLIAM J. GIES.) 1930: pp. 689; 6⅝ x 4 in.; 32 illustrations; $4.00. Ginn and Company, Boston, Mass.

Prosthetic dentistry. An encyclopedia of full and partial denture prosthesis. By IRA GOODSELL NICHOLS, D.D.S., Lieutenant Commander, DC-V (S), USNR, Special Lecturer and Instructor in Dental Prosthesis, Dental Department, U. S. Naval Medical School, Washington, D. C., with forty-five collaborators. 1930: pp. 687; 8 x 5 in.; 830 illustrations; $12.50. C. V. Mosby Co., St. Louis, Mo.

Personal and community health. By CLAIR ELSMERE TURNER, M.A., Dr.P.H., Profesor of Biology and Public Health, Massachusetts Institute of Technology; formerly Associate Professor of Hygiene, Tufts College Medical and Dental Schools. *Third edition.* 1930: pp. 443; 6½ x 3¾ in.; 62 illustrations; $2.75. C. V. Mosby Co., St. Louis, Mo.

Dietetics and nutrition. By MAUDE A. PERRY, B.S., formerly Director of Dietetics; Michael Reese Hospital, Chicago, Ill., and Montreal General Hospital, Montreal, Can. 1930: pp. 332; 6 x 3¾ in.; no illustrations; $2.50. C. V. Mosby Co., St. Louis, Mo.

Special cavity preparations and casting processes for gold inlays. By HARRY ERNEST BULYEA, D.M.D., Professor of Operative Dentistry, Department of Dentistry, University of Alberta. 1930: pp. 30; 7¼ x 4⅛ in.; 17 illustrations. *Bulletin Number 12*, Canadian Dental Research Foundation, Toronto, Can.

Ultra-violet rays. Their application in the practice of dentistry. By ROBERT J. READE, M.A., M.D., C.M., D.D.S. 1929: pp. 31; 7½ x 4⅛ in.; 6 illustrations. *Bulletin Number 13*, Canadian Dental Research Foundation, Toronto, Can.

Annual report of investigations by the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association. Vol. XVIII; 22 assembled reprints; 1929. American Medical Association, Chicago, Ill.

Annals of the Pickett-Thomson Research Laboratory. V. The pathogenic streptococci; their rôle in human and animal disease (continued). Three parts on the rôle of the streptococci; in oral and dental sepsis; in tonsilitis and pharyngitis; and in puerperal sepsis and septic abortion. By DAVID THOMSON, O.B.E., M.B., Ch.B. (Edin.), D.P.H. (Camb.), Hon. Director, Pickett-Thomson Research Laboratory, St. Paul's Hospital, London; and ROBERT THOMSON, M.B., Ch.B. (Edin.), Pathologist to the Pickett-Thomson Research Laboratory. 1929: pp. 484; 8¾ x 6 in.; 49 illustrations; $10.00. Baillière, Tindall and Cox, London, England.

La pratique stomatologique. VII. Orthodontie. By G. IZARD, Professeur d'Orthodontie à l'Ecole Francaise de Stomatologie; Stomatologiste des Hôpitaux de Paris. 1930: pp. 762; 6¾ x 3⅞ in.; 714 illustrations; broché, 95 fr., relié, 110 fr. Masson et Cie, Paris, France.

Dentures et dentiers complets. By F. ACKERMANN, Ancien chef démonstrateur du Professeur Gysi, Université de Zurich; ancien assistant à l'Institut Dentaire et à la Faculté de Medicine de Genéve. 1930: pp. 538; 7½ x 4⅜ in.; 538 illustrations; broché, 135 fr., relié, 150 fr. Masson et Cie, Paris, France.

Orthodontic laboratory manual. By GEORGE M. ANDERSON, D.D.S., Professor of Orthodontia, Baltimore College of Dental Surgery, University of Maryland, Baltimore. 1930: pp. 56; 7 x 4⅛ in.; 23 illustrations; $2.00. C. V. Mosby Co., St. Louis, Mo.

Proceedings of the Seventh Annual Meeting of the American Association of Dental Schools. By R. S. VINSANT, D.D.S., Secretary-treasurer. 1930: pp. 411; 7 x 4 in.; no illustrations. American Association of Dental Schools, Memphis, Tenn.

INDEX

Volume X: 1930

ABSCESSES: subperiosteal; from mandibular first molar, 763. *See also* Infection

Aciduric bacteria. *See* Bacteria

Actinomyces: in pulp and peridental membrane, 405

Adamantinoma: benign tumor; types, 134

Address, president's: eighth general meeting of International Association for Dental Research; A. D. Black, 368, 385

Advertised claims: dental industrial products; validity as related to five so-called tooth-cleaning fluids, 481

AISENBERG, M. S.: tear in cementum, 599

Alaska: Arctic; Eskimo—nutrition and teeth, 387

ALBRAY, R. A.: Vincent's infection; oral manifestations and treatment, 106

Albuminous matter. *See* Protein

Alveoloclasia. *See* Periodontoclasia

American Association of Dental Schools: scientific proceedings; coördinated sessions with International Association for Dental Research, 271, 372, 382

American Child Health Association: Research Monograph III on dental decay; protested, 497

——: ——; reply to criticism, 655

American Dental Association: *Journal;* misstatement regarding publication of paper by Sherman L. Davis corrected, 779

——: research; active, 547

——: resolutions of protest and correction; Research Monograph III, American Child Health Association, 544

——: Section on Mouth Hygiene and Preventive Dentistry; resolutions of protest and correction on Research Monograph III, American Child Health Association, 545

American Indians: enamel defects, 313, 377

American Society for the Promotion of Children's Dentistry: resolutions of protest and correction; Research Monograph III, American Child Health Association, 546

ANDERSON, B. G.: mottled enamel; occurrence in China, 233, 265, 420

ANDERTON, W. P.: discussion; interrelation of medicine and dentistry, 166

Andrews, Robert Robbin: in memoriam, iv

Ann Arbor Section: International Association for Dental Research; proceedings, 1

Anterior lobe. *See* Pituitary

Antibacterial effects: electrodes; electrosterilization, 385

Apical abscesses. *See* Infection

Apparatus: measurement; expansion in dental plasters and investments, 414

APPLEBAUM, EDMUND: five so-called tooth-cleaning fluids; harmful, 485

APPLETON, J. L. T., JR.: electrodes used in electrosterilization; antibacterial effects, 385

ARNIM, S. S.: colloid content of saliva; determined by electrophoresis, 379

Asgis' report: Carnegie Foundation's study of dental education; additional remarks by W. J. Gies, 23

Attrition: teeth; prehistoric Guam, 472

Autopsy: dental and systemic diseases; correlated, 386

BACTERIA: aciduric; relation to caries, 403

Bacteria: electrosterilization; electrodes, 385
——: periapical infections, 381
See also Infection, Microörganisms
BAGNALL, J. S.: properties of saliva; relation to dental condition in children, 393
Balsam: Koch petrifaction method; dissolved celluloid substitute, 252, 401
BARKER, F. J.: dental caries; relation to enamel lamellæ and interglobular spaces, 249
——: vitamin D power to correct dietary deficiency—bone and tooth, 379
Bates, George Andrew: in memoriam, iv
BECKER, J. ERNESTINE: effects of strontium in diet; structure of teeth, 733
BENEDICT, H. C.: colloid content of saliva; determined by electrophoresis, 379
BERENS, CONRAD: discussion; oral infections in relation to diseases of eye, 713
Bering Sea: North; Eskimo—nutrition and teeth, 387
Betel-nut chewing: prehistoric Guam, 453
Betel staining: teeth; prehistoric Guam, 466
BEUST, T. B.: Vincent's infection; microorganisms and treatment, 97
Bibliography: mottled enamel; complete, 561
——: proposed new dental; importance for research, 114
BLACK, A. D.: International Association for Dental Research; president's address, 368
BLAYNEY, J. R.: pulpless tooth; clinical and laboratory investigation, 425
——: repair of previously diseased root-end; after root-canal treatment, 248
Bleachodent: so-called tooth-cleaning fluid; harmful, 481
Blood calcium. *See* Calcium
Blood counts: periapical infections; effect of treatment, 384
Blood phosphorus. *See* Phosphorus
BLUM, THEODOR: clinical oral surgery; related pathology, 337
BLUMGARTEN, S.: discussion; problems in dental research, 560
BÖDECKER, C. F.: enamel defects; teeth of ancient American Indians, 313, 377
——: five so-called tooth-cleaning fluids; harmful, 485
Bogue, Edward Augustus: in memoriam, iv
Bone: calcification; rachitic and nonrachitic diets, 267
——: deficient diet; corrective power of vitamin D, 379
——: determination of density; transmission of x-rays, 239, 403
——: growth; rôle of anterior lobe of pituitary, 393
——: haemangioma; case record, 445
——: sections; new technic, 400. *See also* Method
——: skeletal development; effects of faulty diets, 281
Books: recently received; this *Journal*, 786
BOULGER, E. P.: implanted root apices; reaction of tissue, 446
Breath: offensive; causes and prevention, 418
BRONNER, F. J.: Class-2 cavities; application of engineering principles, 115
——: discussion; problems in dental research, 557
BROWN, R. K.: evaluation of personalities of senior dental students; rating scale, 271, 372
BRYANT, CARRIE K.: periapical infection; effect of treatment on blood-counts and urinalyses, 384
BUNTING, R. W.: control of dental caries; recent experiments, 374

CAHN, L. R.: hypersensitive tooth; neurohistology of pulp, 333
Calcification: centers in tooth development; spacing, 418
——: tooth and bone; rachitic and nonrachitic diets, 267
Calcium: blood; jaw fractures—effect on healing, 134
——: teeth; lime accretions—prehistoric Guam, 468

Calcium: teeth; not replaceable by strontium, 733
Cancer: lower lip; cases at beginning, 249
Cape Province, South Africa: fossil teeth; shark, 439
Carcinoma: epidermoid; adenoid cystic type, 363
——: metastatic; jaw bone, 247
Caries: children; preliminary reply to Oral Hygiene Committee's criticism of Research Monograph III, American Child Health Association, 655
——: ——; Research Monograph III of American Child Health Association protested by Oral Hygiene Committee, 497
——: enamel defects; ancient American Indians, 313
——: internal secretions; relation to enamel, 215
——: odontoclasia; clinically unrecognized, 416
——: prehistoric Guam, 471
——: pre-school children; Honolulu and rural Oahu, 419, 420
——: prevention, 255, 374, 376
——: relation to enamel lamellae and interglobular spaces, 249
——: research; progress, 374
——: susceptibility and immunity; relation of aciduric bacteria, 403
See also Enamel
Carnegie Foundation: study of dental education; additional remarks by W. J. Gies relating to comment by A. J. Asgis, 23
CARR, M. W.: editor's notes; proceedings of New York Academy of Dentistry, 775
——: medico-dental case records; myocarditis, 675
——: ——; toxemia, 173, 406
CARRABINE, OSCAR: discussion; interrelation of medicine and dentistry, 163
Case records. *See* Medico-dental case records
Castration: effects on dentition, 215
Cavities: Class-2; application of engineering principles, 115
CECIL, R. L.: interrelation of medicine and dentistry, 137; discussion, 170
Celloidin: White method; dissolved celluloid substitute for celloidin, 254
Celluloid: dissolved; preparation of ground sections of bone and tooth, 251, 401
Cementoma: hypercementosis; case report, 342
Cementum: tear, 599
Central nervous system. *See* Nervous system
Cervical enamel line: prehistoric Guam, 455
CHASE, S. W.: dissolved celluloid; ground sections of bone and tooth, 251
——: lymph in dentin and enamel; circulation, 383
——: sections of bone and tooth; new aids to histological technique, 400
Chicago Section: International Association for Dental Research; scientific proceedings, 133, 247, 443
Children: caries; preliminary reply to Oral Hygiene Committee's criticism of Research Monograph III, American Child Health Association, 655
——: ——; Research Monograph III of American Child Health Association protested by Oral Hygiene Committee, 497
——: condition of teeth; relation of saliva, 393
See also Pre-school child
China: mottled enamel; occurrence, 233, 265, 420
Circulation: lymph; dentin and enamel, 383
Claims. *See* Advertised claims
Clasp metals: physical studies, 1
COLLINS, M. L.: discussion; esthetics in denture construction, 586
Colloids: saliva; determined by electrophoresis, 379
Columbia University: Gies Fellowship in Biological Chemistry; report of Committee, 771
Commercial products. *See* Dental industrial products

Cook, T. J.: periapical infection; Gottlieb method of checking efficiency of treatment, 402
Coolidge, E. D.: repair after root resection; infected tooth, 133, 381
Craniometry: prehistoric Guam, 462
Curley, Martin: Vincent's infection; laboratory diagnosis, 591
Cusps: teeth; prehistoric Guam, 456
Cysts: incisor canal; case records, 739
——: jaw; operative technique, 406

Daley, F. H.: Vincent's infection; review, 394
Davis, S. L.: misstatement regarding publication of paper in proceedings of New York Academy of Dentistry; corrected, 779
Decay: teeth. *See* Caries
Deciduous teeth. *See* Teeth
Defects. *See* Enamel
Deformation: teeth; prehistoric Guam, 465
Degenerations: root-end; classification, 426
Delves, Edna: control of dental caries; recent experiments, 374
Dental bibliography. *See* Bibliography
Dental caries. *See* Caries
Dental condition. *See* Teeth
Dental decay. *See* Caries
Dental development. *See* Growth
Dental disease: correlated with systemic, 386
Dental education: Carnegie Foundation's study; additional remarks by W. J. Gies relating to comment by A. J. Asgis, 23
——: personalities; rating scale, 271, 372
——: two-three-graduate plan; quotations, 30
Dental enamel. *See* Enamel
Dental granulomata: epithelium, 323, 401
Dental index: prehistoric Guam, 462
Dental industrial products: advertised claims; validity as related to five so-called tooth-cleaning fluids, 481
Dental investments. *See* Investments
Dental lamina: relation to tooth germs, 408
Dental morphology: prehistoric Guam, 415, 454
Dental pathology: prehistoric Guam, 415, 465
Dental plasters. *See* Plasters
Dental pulp. *See* Tooth
Dental research. *See* Research
Dental (stomatological) societies: proceedings. *See* American Association of Dental Schools, American Dental Association, American Society for the Promotion of Children's Dentistry, First District Dental Society of the State of New York, International Association for Dental Research, New York Academy of Dentistry, Oral Hygiene Committee of Greater New York, William Jarvie Society for Dental Research
Dental students. *See* Dental education
Dentin: lymph; circulation, 383
Dentistry: medicine; interrelation, 137
——: not recognized as "scientific;" stated reason, 402
——: operative; "proper materials properly used," 554
——: prosthetic; denture construction, 569
——: status; comment, 23
——: "stomatology;" synonym, iii
Dentition: diet; recommended, 396
——: effects of faulty diet, 281
——: effects of feeding glandular tissues, 215
——: effects of parathyroidectomy and castration, 215
See also Teeth
Dentomucoid: glycoprotein in teeth, 215
Denture construction: esthetics, 576
——: pressure balance, 569
See also Masticatory stress
Development: tooth; spacing of calcification centers, 418
Diagnosis: Vincent's infection; laboratory, 591
Diet: calcium replaced by strontium; effects on teeth, 733
——: control of caries, 374
——: deficient in mineral salts; structural changes in tooth and jaw, 378

Diet: dentition, 396
——: Eskimo; teeth, 387
——: faulty; effects on bone and tooth, 281
——: food complex; prehistoric Guam, 452
——: mouth flora; effects, 33
——: rachitic and non-rachitic; calcification of bone and tooth, 267
——: rickets; effects of "Vigantol" on teeth, 411
——: saliva; pH values, 84
——: vitamin C deficiency; histological effects on teeth, 407
——: vitamin D; power to correct dietary deficiency, 379
——: vitamin D ("Vigantol"); histological effects on teeth and jaw, 408
Discussion: dental research; problems, 556
——: medicine and dentistry; interrelation, 157
——: oral infections; relation to diseases of eye, 713
——: prosthetic dentistry; denture construction, 582
Diseases: dental and systemic; correlation, 219, 386
——: eye; relation of oral infections, 685
——: inflammatory, in jaws from teeth; indications for surgical treatment, 412
See also names of diseases
Downs, W. G., Jr.: anterior lobe of pituitary in growth changes; bones and teeth, 109, 393, 601
——: dental and systemic diseases; correlated at autopsy, 386
Dunning, H. S.: discussion; interrelation of medicine and dentistry, 169
——: oral infections; relation to diseases of eye, 706
Dunning, W. B.: discussion; interrelation of medicine and dentistry, 158
Dwyer, H. S.: protest; Research Monograph III, American Child Health Association, 500, 536
Dwyer, J. G.: discussion; oral infections in relation to diseases of eye, 717

EDENTULOUS areas: soft tissues on ridge; effects of masticatory stress, 386
Education. *See* Dental education
Ekay: so-called tooth-cleaning fluid; harmful, 481
Electrodes: electrosterilization; anti-bacterial effects, 385
Electrophoresis. *See* Method
Electrosterilization: electrodes; antibacterial effects, 385
Enamel: caries; relation to enamel lamellae and interglobular spaces, 249
——: ——; relation of internal secretions, 215
——: cervical line; prehistoric Guam, 455
——: defects; ancient American Indians, 313, 377
——: ——; fissures and pits—review, 659
——: development; diffusion of substances from blood, 215
——: horizontal grooves; prehistoric Guam, 455
——: lymph; no circulation, 211, 383, 419
——: mottled; occurrence in China, 233, 265, 420
——: ——; review, 268, 561
——: Nasmyth's membrane; structure and formation, 443
——: permeability; quantitative determination, 447, 727
——: ——; substances in blood, 215
——: ——; substances in saliva, 215
——: ——; water and salts, 377
——: protein; keratin, 187
——: ——; no mucoid or nucleoprotein, 418
——: ——; no soluble protein, 195, 418
——: ——; identification, 268
Endowment Fund: *Journal of Dental Research;* report, iii
Engineering principles: applied to cavities; Class 2, 115
Epidermoid carcinoma. *See* Carcinoma
Epithelial attachment: tooth; shifting, 445
Epithelium: dental granulomata, 323, 401
Eskimo: nutrition; teeth, 387
Essig, Norman: denture construction; pressure balance, 569; discussion, 588
Esthetics: denture construction, 576

Ethnic deformation: teeth; prehistoric Guam, 465
Expansion: dental plasters and investments; apparatus for measurement, 414
Ex-Tartar: so-called tooth-cleaning fluid; harmful, 481
Eye: diseases; relation of oral infections, 685

FELLOWSHIP: Gies, in Biological Chemistry; report of Committee, 771
FERRIS, H. C.: discussion; problems in dental research, 556, 559
First District Dental Society, State of New York: scientific proceedings; footnote references, 97, 337, 425, 547, 763
First molar: enamel defects, 497, 659
——: mandibular; subperiosteal abscesses, 763
Fissures: enamel; review, 659
FLEMING, W. C.: mouth flora under different dietary conditions; quantitative examination, 33
Flora. *See* Mouth flora
Focal infection. *See* Infection
Food. *See* Diet
Fossil teeth. *See* Teeth
Fractures. *See* Jaw fractures
FRANKE, ELIZABETH C.: dental enamel; permeable to substances in saliva, 215
Fu ., Endowment: *Journal of Dental Research;* report, iii

GASSERIAN ganglion: lues; central nervous system, 405
Geographical distribution: mottled enamel, 268, 420, 561
Giant-cell tumor: case report, 354
Gies Fellowship in Biological Chemistry: Columbia University; papers by Fellows, 7, 187, 268, 403
——: ——; report by Fellowship Committee, 771
GIES, W. J.: Carnegie Foundation's study of dental education; additional remarks on a reference by A. J. Asgis, 23
GIES, W. J.: dental caries; relation of internal secretions to development and condition of enamel, 215
——: dentomucoid; none in enamel, 418
——: findings of Research Council of New York Academy of Dentistry; Bleachodent, Ekay, Ex-Tartar, Snovy-White, and Tartaroff—harmful, 481
——: International Association for Dental Research; proceedings of eighth general meeting, 367
——: ——; Secretary's notes on proceedings, 782
——: mucoid absent from enamel, 418
——: no soluble protein in enamel, 418
——: nucleoprotein absent from enamel, 418
——: protest; Research Monograph III, American Child Health Association, 500, 536
——: this Index, 791
GILLETT, H. W.: discussion; interrelation of medicine and dentistry, 157
——: oral infections; relation to diseases of eye, 694
Gingiva: normal; relation to pathology, 250. *See also* Periodontoclasia
Glandular tissues: feeding; effects on dentition, 215
Glycoprotein: teeth; dentomucoid, 215
——: none in enamel, 418
Gnathic index: prehistoric Guam, 464
GODSOE, W. H.: unusual cases; dental infirmary of University of Toronto, 123
Gold-alloy restorations: procedures and materials, 548
Gottlieb method. *See* Method
Granulomata. *See* Dental granulomata
Graphic registration. *See* Method
GRIEVE, G. W.: science of orthodontia; theories that obstruct its progress, 395
Grieves, Clarence Jones: in memoriam, iv
GROSSMAN, L. J.: electrodes for electrosterilization; antibacterial effects, 385
Growth: bones and teeth; faulty diets, 281
——: tadpoles; pituitary extract, 109
——: teeth and other tissues; anterior lobe of pituitary, 393, 601

Guam: prehistoric; dental morphology and pathology, 415, 451
——: ——; food complex, 452
Gum. *See* Gingiva

HAEMANGIOMA: bone; case record, 445
Halitosis: causes and prevention, 418
HARD, DOROTHY G.: control of dental caries; recent experiments, 374
HARDY, I. R.: denture construction; esthetics, 576; discussion, 589
HATTON, E. H.: actinomyces and actinomyces-like organisms; pulp and peridental membrane, 405
HAYHURST, T. E.: instruments; angle of sharpening to insure efficiency, 398
HAYWARD, S. S.: fossil teeth of sharks; Cape Province, South Africa, 439
Healing process: jaw fractures; conditions, 134
Health problems: pulpless tooth; treatment, 427
HEFT, HATTIE L.: Do substances in blood pass from pulp into enamel?, 215
Herpes simplex: mouth manifestations, 228
Hexylresorcinol: control of caries; research, 374
HILL, T. J.: dental granulomata; epithelium, 323, 401
HIRSCHFELD, ISADOR: migrating teeth; relationship of inflammatory tissue in pyorrheal pockets, 382
——: periodontal disease; diagrammatic record, 266
——: Vincent's infection; clinical aspects and treatment, 107
HOFER, OTTO: cysts in jaws; operative technique, 406
Honolulu: pre-school child; odontoclasia, 416
HOSKINS, MARGARET M.: parathyroidectomy; effect on teeth, 121
HOWITT, BEATRICE F.: mouth flora under different dietary conditions; quantitative examination, 33
HYATT, T. P.: enamel pits and fissures; review, 659
HYATT, T. P.: protest; Research Monograph III, American Child Health Association, 500, 536
Hypercementosis: cementoma; case report, 342
Hypersensitive tooth. *See* Tooth
Hypophysis. *See* Pituitary

IMPACTION: root; absorption at neck, 338
Implantation: root apices; tissue reactions, 446
Impressions: dimensions; variations, 550
Incisor: upper; apparent shortening, 135
Incisor canal: cysts; case records, 739
Indians. *See* American Indians
Industrial products. *See* Dental industrial products
Infection: focal; experimentally produced, 399
——: implanted root apices; tissue reactions, 446
——: jaw fractures; effect on healing, 134
——: oral; relation to diseases of eye, 685
——: periapical; bacteria, 381
——: ——; blood counts and urinalysis before and after treatment, 384
——: ——; Gottlieb method of checking treatment, 402
——: residual; correlation of radiographic and histologic findings, 113, 394
——: root resection; repair, 133
——: Vincent's; laboratory diagnosis, 591
——: ——; oral manifestations and treatment, 97
——: ——; review, 394
See also Root-canal treatment
Inflammatory diseases. *See* Diseases
Inflammatory tissue: pyorrheal pockets; relation to migrating teeth, 382
Inlay technics: criticism, 555
Inorganic salts: metabolism. *See* Metabolism
INOUYE, J. M.: Gies Fellow in Biological Chemistry; Columbia University, 7, 771
——: salivary mucin; biochemical data, 7
Instruments: efficient; angle of sharpening, 398

Internal secretions: relation to enamel and caries, 215
International Association for Dental Research: lists of members and officers, 784
——: new members and officers (1930), 420
——: president's address, 368, 385
——: proceedings of *Association;* eighth general meeting (1930), 367, 782
——: ——; footnote references, 33, 173, 187, 219, 233, 239, 251, 255, 271, 281, 313, 323, 451, 561, 675
——: ——; secretary's notes, 782
——: proceedings of *Sections*; *Ann Arbor*, 1
——: ——; *Chicago*, 133, 247, 443
——: ——; *New York*, 113, 265, 420
——: sessions with the American Association of Dental Schools, 271, 372, 382
Investments: expansion; apparatus for measurement, 414

JACKSON, Holmes Condict: in memoriam, iv
Jarvie Society. *See* William Jarvie Society
Jarvie, William: in memoriam, iv
Jaw: bone; metastatic carcinoma, 247
——: cysts; operative technique, 406
——: fractures; healing as affected by various conditions, 134
——: inflammatory diseases from teeth; indications for surgical treatment, 412
——: movements; graphic registration, 404
——: osteodystrophia fibrosa localisata; symptoms and diagnosis, 409
——: soft tissues on edentulous areas; effects of masticatory stress, 386
——: structural changes; diets deficient in mineral salts, 378
——: synthetic vitamin D; histological findings, 408
——: tumors; adamantinoma, 134
Jenkins, Newell Sill: in memoriam, iv
JONES, MARTHA R.: dental findings; preschool children on Island of Oahu, 419, 420.
——: inorganic salt metabolism; effect of faulty diet on bone and tooth, 281
JONES, MARTHA R.: odontoclasia; clinically unrecognized tooth decay in pre-school child of Honolulu, 416
JOSEPH, T. V.: discussion; problems in dental research, 558, 559
Journal of Dental Research: books recently received, 784
——: Endowment Fund; report, iii
——: misstatement regarding publication of paper by Sherman L. Davis; corrected, 779
Journal of the American Dental Association: misstatement regarding publication of paper by Sherman L. Davis in proceedings of New York Academy of Dentistry; corrected, 779

KARSHAN, MAXWELL: calcification of bones and teeth; rachitic and non-rachitic diets, 267, 420
——: five so-called tooth-cleaning fluids; harmful, 485
——: keratin; chemistry and staining reactions, 181
KENNEDY, EDWARD: discussion; prosthetic dentistry, 587
Keratin: chemistry and staining reactions, 181
——: dental enamel; qualities, 187
KLEIN, HENRY: effects of strontium in diet on structure of teeth, 733
——: lead sector-disk; density of bone and tooth structure expressed in transmission of x-rays, 239, 403
——: permeability of enamel; quantitative determinations, 447, 727
Koch petrifaction method. *See* Method
KOTANYI, EMERICH: experimental scurvy; histological effects on teeth, 407
KRONFELD, RUDOLF: adamantinoma; types, 134
——: cancer of lower lip; cases at beginning, 249
——: Nasmyth's membrane; structure and formation, 443
——: vitamin D; power to correct dietary deficiency—bone and tooth, 379

LARSEN, N. P.: dental findings; pre-school children on Island of Oahu, 419, 420
——: odontoclasia; clinically unrecognized tooth decay in pre-school child of Honolulu, 416
Lead sector-disk: determination of density of bone and tooth; transmission of x-rays, 239, 403
LEAK, W. H.: discussion; problems in dental research, 558
LEIGH, R. W.: prehistoric Guam; dental morphology and pathology, 415, 451
LEONARD, H. J.: discussion; interrelation of medicine and dentistry, 166
Lichen planus: mouth manifestations, 220
Light: measure; astronomical distances, 4
Lime accretions: dental; prehistoric Guam, 468
Lip: lower; cases of beginning cancer, 249
LITTEN, A. S.: discussion; problems in dental research, 556
Lues: central nervous system; gasserian ganglion, 405. *See also* Syphilis
LUNDQUIST, G. R.: pyorrhea as clinician sees it, 444
Lymph: circulation; dentin and enamel, 383

MACMILLAN, H. W.: subperiosteal abscesses; from mandibular first molar, 763
Mandible: prehistoric Guam, 465
Mandibular first molar. *See* First molar
Marginal ridges: incisors; prehistoric Guam, 456
Masticatory stress: effects on soft tissue; edentulous areas of ridge, 386
Maxillo-alveolar index: prehistoric Guam, 463
MCCALL, J. O.: discussion; interrelation of medicine and dentistry, 159
——: ——; oral infections in relation to diseases of eye, 723
——: prevention; caries and periodontoclasia, 255, 376
——: residual infection; radiographic and histologic findings correlated, 113, 394
MCCOLLUM, E. V.: calcium replaced by strontium in diet; effects on structure of teeth, 733
MCEVOY, ELMER: Vincent's infection; laboratory diagnosis, 591
MCFALL, T. A.: enamel; permeability to water and salts, 377
MCKAY, F. S.: mottled enamel; review of cause and geographical distribution, 268, 420, 561
Medicine: dentistry; interrelation, 137
Medico-dental case records: abscesses; subperiosteal, 763
——: clinical oral surgery, 337
——: cysts; incisor canal, 739
——: haemangioma, 445
——: myocarditis, 675
——: skin diseases; primary mouth manifestations, 219, 269
——: toxemia, 173, 406
See also Infection
Meltzer, Samuel James: in memoriam, iv
Memorial: Andrews, Bates, Bogue, Grieves, Jackson, Jarvie, Jenkins, Meltzer, Peeso, Van Woert, iv
——: Bissell B. Palmer, Sr.; resolution by New York Academy of Dentistry, 780
Mental foramen: prehistoric Guam, 461
MERRITT, A. H.: discussion; oral infections in relation to diseases of eye, 719
Metabolism: inorganic salt; effects of faulty diet on bone and tooth, 281. *See also* Nutrition
Metals. *See* Clasp metals
Metastatic carcinoma. *See* Carcinoma
Method: dental and systemic diseases; correlated, 386
——: determination; density of bone and tooth to x-rays, 239
——: diagrammatic record; periodontal disease, 266
——: electrosterilization; electrodes, 385
——: enamel proteins. *See* "protein in enamel" below
——: gold-alloy restorations, 548
——: Gottlieb; check on efficiency of treatment of periapical infection, 402

Method: graphic registration; jaw movements, 404
——: ground sections of bone and tooth; dissolved celluloid, 251, 401
——: ——; Koch petrifaction, 252, 401
——: ——; new aids to histological technique, 400
——: ——; White celloidin, 254, 401
——: operative dentistry; deficiencies, 547
——: operative technic; cysts of jaw, 406
——: protein in enamel; separation, identification, and determination, 187
——: ——; demonstration of methods, of separation and identification, 268
——: quantitative; enamel permeability, 447, 727
——: ——; mouth flora, 39
——: ——; salivary mucin, 7
——: root-canal technique; cultural control, 419
——: saliva; determination of colloid by electrophoresis, 379
See also Apparatus
Microörganisms: actinomyces and actinomyces-like; in pulp and peridental membrane, 405. *See also* Bacteria, Mouth flora
Migrating teeth. *See* Teeth
MILLER, E. G., Jr.: dentition; effects of feeding glandular tissues, 215
——: ——; effects of parathyroidectomy and castration, 215
——: Gies Fellowship in Biological Chemistry; report by Fellowship Committee, 771
MILLER, H. A.: dental periapical infections; bacteria, 381
MINER, L. M. S.: interrelation of medicine and dentistry, 150; discussion, 170
Mineral salts: metabolism. *See* Metabolism
Models: dimensions; variations, 550
Molar tooth. *See* Teeth
Monograph III: American Child Health Association; protested, 497
——: ——; preliminary reply to Oral Hygiene Committee's criticism, 655
MOON, V. H.: chronic focal infection; experimentally produced, 399
MOOREHEAD, F. B.: bone haemangioma; case record, 445
Mottled enamel. *See* Enamel
Mouth flora: quantitative examination; various diets, 33
Mouth manifestations: skin diseases, 219, 269
Mucin. *See* Saliva
Mucinate. *See* Saliva
Mucoid: teeth; none in enamel, 418
Myocarditis: medico-dental case record, 675

NASMYTH'S membrane: enamel; structure and formation, 443
Naso-palatine (incisor) canal: cysts; case records, 739
NEERGAARD, A. E.: discussion; interrelation between medicine and dentistry, 163
Nervous system: lues; gasserian ganglion, 405
NEUSTADT, EGON: tooth eruption; related to plane of occlusion, 120
New York Academy of Dentistry: editor's notes on proceedings, 775
——: joint session; Section on Ophthalmology, New York Academy of Medicine, 685
——: list of officers and committees, 775
——: memorial resolution; Bissell B. Palmer, Sr., 780
——: paper by Sherman L. Davis; misstatement about publication in proceedings corrected, 779
——: Research Council; findings on five so-called tooth-cleaning fluids, 481
——: scientific proceedings; footnote references, 481, 569, 685
New York Academy of Medicine: Section on Ophthalmology; joint session with New York Academy of Dentistry, 685
New York Section: International Association for Dental Research; scientific proceedings, 113, 265, 420

NOLAND, LEILA: glycoprotein in teeth; dentomucoid, 215
Nucleoprotein: teeth; none in enamel, 419
Nutrition: teeth; Eskimo, 387. *See also* Diet, Metabolism

OAHU: Island; dental findings in preschool children, 419, 420
Occlusion: normal; dangers during shedding of deciduous molars, 409
——: plane; relation of tooth eruption, 120
——: teeth; prehistoric Guam, 460
Odontoclasia: dental decay; clinically unrecognized, 416
Odontoma: case report, 347
Operative dentistry: "proper materials properly used," 554
——: inlay technics; criticism, 555
Operative technique. *See* Method
Oral Hygiene Committee of Greater New York: Research Monograph III of American Child Health Association; protest including report by sub-committee, 497
——: ——; reply by Director of Research of American Child Health Association, 655
——: scientific proceedings; footnote references, 500, 536, 655, 659
Oral infections. *See* Infection
Oral manifestations. *See* Mouth manifestations
Oral microörganisms. *See* Microörganisms
Oral surgery: clinical; related pathology, 337. *See also* Jaw
Oral symptoms: skin diseases, 219
ORBAN, BALINT: relation between dental lamina and tooth germs, 408
Orthodontia: science; theories that obstruct its progress, 395
Osteitis. *See* Osteodystrophia, Periapical osteitis
Osteodystrophia fibrosa localisata: jaws; symptoms and diagnosis, 409
Osteogenic sarcoma: case report, 357
OWRE, ALFRED: discussion; interrelation of medicine and dentistry, 161

PALMER, B. B., JR.: editor; scientific proceedings of New York Section, International Association for Dental Research, 113, 265
——: medico-dental case record; myocarditis, 675
——: ——; toxemia, 173, 406
Palmer, B. B., Sr.: memorial resolution; New York Academy of Dentistry, 780
PALMER, G. T.: Oral Hygiene Committee's criticism of American Child Health Association's Research Monograph III; preliminary reply, 655
Papilla palatina: cysts; case records, 739
Paradental lesions: teeth; prehistoric Guam, 474. *See also* Periodontoclasia
Parathyroidectomy: effect on teeth, 121, 215
Pedo-periodontoclasia: destructive; chronic, 123
Peeso, Frederick Austin: in memoriam, iv
Pemphigus: mouth manifestations, 225
PENTZ, W. R.: gasserian ganglion; lues of central nervous system, 405
Periapical infection. *See* Infection
Periapical osteitis: teeth; prehistoric Guam, 473
Peridental membrane: actinomyces and actinomyces-like organisms, 405
Periodontoclasia: diagrammatic record, 266
——: pedo-; destructive chronic, 123
——: prehistoric Guam, 469
——: prevention, 255, 376
See also Paradental lesions, Pyorrhea
Permeability: enamel; quantitative determinations, 447, 727
Personalities: dental students; rating scale, 271, 372
PHILLIPS, G. P.: movements of jaw; graphic registrations, 404
Phosphorus: blood; jaw fractures—effect on healing, 134
PIGOTT, J. P.: dental and systemic diseases; correlated at autopsy, 386
——: root-canal technique; cultural control, 419
Pits: enamel; review. 659

Pituitary: anterior lobe; growth changes—bone and tooth, 393, 601
——: extracts; growth of tadpoles, 109
Plasters: expansion; apparatus for measurement, 414
POLEVITZKY, K.: periapical infection; effect of treatment on blood-counts and urinalyses, 384
Prehistoric dental conditions. *See* Guam
Pre-school child: dental findings; Island of Oahu, 419, 420
——: odontoclasia; clinically unrecognized, 416
Pressure balance: denture construction, 569
Prevention: caries and periodontoclasia, 255, 376
——: dental caries; recent research, 374
PRINZ, HERMANN: offensive breath; causes and prevention, 418
PRITCHARD, G. P.: dental findings; pre-school children on Island of Oahu, 419, 420
——: odontoclasia; clinically unrecognized tooth decay in pre-school child of Honolulu, 416
Proceedings: dental (stomatological) societies. *See* Dental (stomatological) societies
"Proper materials: properly used;" operative dentistry, 554
Prosthetic dentistry: denture construction, 569. *See also* Jaw
Protein: dental enamel; keratin, 187
——: ——; no mucoid or nucleoprotein, 418
——: ——; no soluble protein, 195, 418
——: ——; quantitative determination, 207
——: ——; separation and identification, 268
See also Keratin
Public health: aspects of dental decay; children, 497, 655
Pulp: actinomyces and actinomyces-like organisms, 405
——: hypersensitive tooth; neuro-histology, 333
See also Teeth
Pulpless tooth. *See* Teeth
Pyorrhea: clinical conditions, 444
——: pockets—inflammatory tissue; relationship to migrating teeth, 382
See also Periodontoclasia

RACHITIC diet. *See* Diet.
Rating scale: personalities; dental students, 271, 372
Rays. *See* Ultraviolet rays, X-rays
READE, R. J.: ultraviolet rays; review, 397
Repair: diseased root-end; after root-canal treatment, 248
——: resection of root; after chronic infection, 133, 381
Research: American Dental Association; active, 547
——: dental program; presidential address, 368
——: *Journal of Dental Research;* report on Endowment Fund, iii
——: problems; operative dentistry, 547
See also International Association for Dental Research, Research Council, Research Monograph
Research Council: New York Academy of Dentistry; findings on five so-called tooth-cleaning fluids, 481
Research Monograph III: American Child Health Association; protested, 497
——: ——; defended, 655
Residual infection. *See* Infection
Resolutions: in memoriam; Bissell B. Palmer, Sr.—New York Academy of Dentistry, 780
——: protest and correction; Research Monograph III of American Child Health Association, 544
Resorption: deciduous teeth; findings, 443
Restorations: gold-alloy; procedures and materials, 548
RICKERT, U. G.: editor; proceedings of Ann Arbor Section, International Association for Dental Research, 1
Rickets. *See* Diet
Ridge. *See* Jaw, Teeth

RITCHIE, S. G.: sensitive apparatus; measurement of expansion in dental plasters and investments, 414
Root: absorption at neck; impaction, 338
——: apices implanted; tissue reactions, 446
——: canal treatment; repair after root resection, 381
——: ——; repair of diseased root-end, 248
——: end; degenerations—classification, 426
——: ——; repair after root-canal treatment, 248
——: resection; repair after canal filling, 381
——: ——; repair after chronic infection, 133
ROSEBURY, THEODOR: aciduric bacteria; relation to susceptibility and immunity to caries, 403
——: enamel proteins; separation, identification, and determination, 187
——: ——; methods for separation and identification, 268
——: Gies Fellow in Biological Chemistry; Columbia University, 187, 268, 403, 771
Ross, Victor: Gies Fellow in Biological Chemistry; Columbia University, 772
RUFUS, W. C.: light; "news from the sky," 4

SALIVA: colloid; determined by electrophoresis, 379
——: constituents; diffusion into enamel, 215
——: mucin; biochemical data, 7
——: ——; submaxillary—biochemical data, 8
——: mucin content; determination, 16
——: mucinate, 7, 8
——: pH determinations; different diets, 84
——: properties; relation to dental condition in children, 393
Salts, mineral: deficient in diet; structural changes in jaw and tooth, 378
Sarcoma: osteogenic; case report, 357
SCHÖNBAUER, FRANZ: jaws and teeth of child treated with "Vigantol" (synthetic vitamin D); histological findings, 408
School Health Research Monograph III. *See* Monograph III
SCHROFF, JOSEPH: cysts in incisor canal; naso-palatine (incisor) canal and papilla palatina, 739
——: editor's notes on proceedings of William Jarvie Society for Dental Research, 781
——: skin diseases; primary mouth manifestations, 219, 269, 420
SCHWARZ, A. M.: normal occlusion; dangers during shedding of deciduous molars, 409
SCOTT, E. O.: clasp metals; physical studies, 1
Scurvy: teeth; histological findings, 407
Sections: bone and teeth. *See* Method
Sector disk. *See* Lead sector-disk
Shark: fossil teeth; Cape Province, South Africa, 439
SHAW, J. C. M.: fossil teeth of sharks; Cape Province, South Africa, 439
SHEINMAN, KANNON: protest; Research Monograph III, American Child Health Association, 500, 536
Skeletal development: effects of faulty diets, 281. *See also* Bone
SKILLEN, W. G.: gingiva; relation of normal conditions to pathology, 250
——: metastatic carcinoma; jaw bone, 247
——: shifting epithelial attachment, 445
Skin diseases: primary oral manifestations, 219, 269, 420
Snovy-White: so-called tooth-cleaning fluid; harmful, 481
Societies: dental (stomatological); proceedings. *See* Dental (stomatological) societies
SOUDER, WILMER: dental research; problems, 547; discussion, 556, 557, 558, 559, 560
SPRING, KARL: osteodystrophia (osteitis) fibrosa localisata of jaw; symptoms and diagnosis, 409
STEINBUGLER, W. F. C.: oral infections; relation to diseases of eye, 685
Sterilization. *See* Electrosterilization
STEVENSON, P. H.: mottled enamel; occurrence in China, 233, 265, 420

STEWART, G. D.: discussion; interrelation of medicine and dentistry, 168
STILLMAN, P. R.: caries and periodontoclasia; prevention, 255, 376
Stomatological societies: proceedings. *See* Dental (stomatological) societies
Stomatology: proposed substitute for "dentistry;" discussed, 29
——: ——; synonym of "dentistry," iii
Strontium: substituted for calcium in food; effects on teeth, 733
Structure. *See* Enamel, Jaw, Teeth
Submaxillary mucin. *See* Saliva
Subperiosteal abscesses. *See* Abscesses
Surgical treatment. *See* Technique, Treatment
Systemic diseases. *See* Diseases
SWANSON, J. H.: tooth development; spacing of calcification centers, 418
Syphilis: jaw fractures; effect on healing, 134. *See also* Lues

TABER, L. B.: Vincent's infection; laboratory diagnosis, 591
Tartaroff: so-called tooth-cleaning fluid; harmful, 481
Technique: operative; cysts in jaws, 406
——: operative dentistry; deficiencies, 547. *See also* Method
Teeth (tooth): attrition; prehistoric Guam, 472
——: atypical; prehistoric Guam, 458
——: calcification; rachitic and non-rachitic diets, 267
——: children; relation of saliva, 393. *See also* "pre-school children," below
——: "cleaning" fluids—Bleachodent, Ekay, Ex-Tartar, Snovy-White, Tartaroff; harmful, 481
——: deciduous; resorption, 445
——: determination of density; transmission of x-rays, 239, 403
——: development; spacing of calcification centers, 418
——: dietary deficiency; calcium replaced by strontium, 733
——: ——; corrective power of vitamin D, 379
Teeth (tooth): dietary deficiency; structural changes, lack of mineral salts, 378
——: effect of parathyroidectomy, 121
——: epithelial attachment; shifting, 445
——: eruption; related to plane of occlusion, 120
——: Eskimo; nutrition, 387
——: fossil shark; Cape Province, South Africa, 439
——: germs; relation to dental lamina, 408
——: growth; rôle of anterior lobe of pituitary, 393, 601
——: Guam; prehistoric conditions, 451
——: hypersensitive; neuro-histology of pulp, 333
——: incisor; apparent shortening (upper), 135
——: ——; marginal ridge, prehistoric Guam, 456
——: migrating; relationship to inflammatory tissue in pyorrheal pockets, 382
——: mucoid; none in enamel, 418
——: nucleoprotein; none in enamel, 418
——: occlusion; dangers during shedding of deciduous molars, 409
——: prehistoric conditions; Guam, 451
——: pre-school children; Honolulu and rural Oahu, 419, 420
——: pulpless; clinical and laboratory data, 425
——: related inflammatory diseases in jaws; indications for surgical treatment, 412
——: rickets; effects of "Vigantol," 411
——: scurvy; histological findings, 407
——: sections; new technic, 400. *See also* Method
——: synthetic vitamin D; histological findings, 408
——: "Vigantol;" histological findings, 408
See also Enamel, Dentition, Pulp, Root
TENCH, R. W.: discussion; pressure balance in denture construction, 582
TISDALL, F. F.: diet and dentition, 396
Tissues: growth effects; anterior lobe of pituitary, 601
——: reaction; implanted root apices, 446
See also Edentulous areas

Tooth. *See* Teeth
Toxemia: medico-dental case record, 174, 406
TRACY, W. D.: discussion; interrelation of medicine and dentistry, 164
Treatment: periapical infection; blood-counts and urinalyses, 384
——: ——; Gottlieb method of checking efficiency, 402
——: inflammatory disease in jaw; from teeth, 412
——: pulpless tooth; successful, 425
——: Vincent's infection. *See* Infection
Tumor: giant-cell; case report, 354. *See also* Adamantinoma
Two-three-graduate plan. *See* Dental education
TYLMAN, S. D.: editor; scientific proceedings of Chicago Section, International Association for Dental Research, 133, 247, 443

ULTRAVIOLET rays: review, 397
University of Toronto; dental infirmary; unusual cases, 123
URBAN, L. B.: deciduous teeth; findings on resorption, 445
Urinalyses: periapical infections; effect of treatment, 384

VAN DEN BERG, E. J.: discussion; prosthetic dentistry, 588
Van Woert, Frank Thorn: in memoriam, iv
"Vigantol:" synthetic vitamin D; histological effects on jaws and teeth, 408
——: ——; rickets—effects on teeth, 411
Vincent's infection. *See* Infection
Vitamin: deficiency; effects on bone and tooth, 379
Vitamin C: deficiency; histological effects on teeth, 407
Vitamin D: power to correct dietary deficiency; bone and tooth, 379. *See also* "Vigantol"

WADDELL, R. W.: discussion; interrelation of medicine and dentistry, 159
WAGENER, EDNA H.: Vincent's infection; laboratory diagnosis, 591
WARD, M. L.: clasp metals; physical studies, 1
WAUGH, L. M.: Eskimo of North Bering Sea and Arctic Alaska; nutrition and teeth, 387
WEINBERGER, B. W.: dentistry; not recognized as "scientific," 402
——: proposed new dental bibliography; stated importance for research, 114
WEINMANN, JOSEF: rickets; effects of "Vigantol" on teeth, 411
White method. *See* Method
William Jarvie Society for Dental Research: editor's notes on proceedings, 781
——: scientific proceedings; footnote references, 739
William J. Gies Fellowship. *See* Gies Fellowship
WILLIAM, MAURICE: protest; Research Monograph III, American Child Health Association, 500, 536
WILLMAN, WARREN: upper incisor; apparent shortening, 135
WOLF, HERMANN: inflammatory diseases of jaws arising from teeth; indications for surgical treatment, 412
WOLFE, J. J.: diet deficient in mineral salts; structural changes in jaw and tooth, 378
WOOD, F. C.: discussion; interrelation of medicine and dentistry, 166
WOODARD, D. E.: healing process in jaw fractures; relation of infection, delay in reduction, syphilis, and blood calcium and phosphorus content, 134
WRIGHT, W. H.: masticatory stress; effects on soft tissues in edentulous areas of ridge, 386

X-RAYS: percentage transmission through bone and tooth; lead sector-disk for determination, 239, 403

YOUNG, E. G.: saliva; relation to dental condition in children, 393

Quotations Relating to "Dentistry or Stomatology"

THIS JOURNAL uses the term "dentistry" to signify the science and art of oral health service in all its local and systemic relationships. The meanings of words, which are not necessarily restricted to *etymological* significance, acquire expanded or new import in accordance with convenience and usage. Thus the term "medicine," which originally meant to remedy or cure, now also means *to make remedial or curative treatment unnecessary.* "Stomatology" is a synonym for "dentistry." We prefer the older term because its use respects both the history of dentistry and the efforts of those who have brought dentistry to its present status.—*Jour. of Dental Research*, 1929, ix, p. iii.

If dentistry were merely dental mechanics, a two-year course in dental technology, based on graduation from a high school and conducted along lines similar to those for the education of optometrists, would probably afford sufficient training for dental practice, and oral health-service could be reserved for a new specialty to include oral surgery, which might be named stomatology and made analogous to ophthalmology and other specialties of medicine. But with dentistry developed into the *full service equivalence* of an oral specialty of medicine, the creation of a medical specialty of stomatology would be redundant—and there would be no occasion to substitute "stomatology" for the name established by custom.—*Carnegie Foundation's Bulletin on Dental Education*, 1926, p. 135.

The assumption that dentistry would be something more or better than it is, if all or a part of it were called "stomatology," exemplifies a readiness to attach more importance to labels than to contents, or more significance to make-believe than to reality. The term "medicine" originally meant to remedy or cure. ["*Medicina* (ars)"—healing art; "*medicina* (res)"—a remedy; "*medicus*"—of healing; also a physician.] Today "medicine," by including measures to *prevent* sickness and by throwing *etymology* overboard, means not only to remedy or cure, but also *quite the contrary*—to make remedial or curative treatment unnecessary. As medical knowledge has advanced and medical procedures have been changed in consonance, including the development of prevention and of surgery, new meanings have been imparted to the term "medicine." But instead of replacing "medicine" by a succession of words intended to indicate *etymologically* the broader or deeper modern meanings, the import of the original term has been conveniently enlarged as the scope of medical practice has expanded. Thus, the term "medicine" has come to mean more and more as *physicians*, without restricting themselves to its *etymology*, have included *more and more in its significance.* Along the same line of reasoning one sees clearly that, although *etymologically* the term "dentistry" refers only to the teeth, nevertheless logically it also comprehends the contiguous parts and their systemic relationships. Certainly by *usage* in conformity with this logic, and also with the criteria of common sense, the term "dentistry" has *acquired* the broader meaning suggested by the more detailed and lengthy term "oral health-service." By logic, usage, and convenience, the term "dentistry" has thus become the *practical* equivalent of "stomatology," which *etymologically* means the sum of scientific knowledge concerning the mouth, but *etymologically does not include the rest of the body* (although "stomatology" is now *used* by some to include systemic relationships with the mouth). This being true, we are unable to agree that "dentistry" and "dentists," which are terms that have long represented the earnest and aspiring endeavors of generations of faithful practitioners of oral health-service, should be formally replaced for *etymological* reasons by "stomatology" and "stomatologists."—*American College of Dentists*, March, 1929.

The New York Academy of Dentistry . . . without any diminution of its abiding purpose to promote intimate association and cooperation between dentists and the practitioners of all other kinds of health-service, believes that, in the interest of the patient, dentistry should be *continued* as a separately organized profession, based on systems of ethics, education, jurisprudence, and journalism that will fully meet all public needs at each stage of its evolution.—*N. Y. Academy of Dentistry, April, 1929.*

The Dental Society of the State of New York . . . expresses the earnest conviction . . . that the most effectual development of oral health-service will be insured by the *continuance* of dentistry as a separately organized profession . . . [and] also records its unabated desire and intention to promote personal and professional accord, cooperation, and mutual helpfulness between dentists and the practitioners of medicine and all other kinds of health-service.—*Dental Society of the State of New York, May, 1929.*

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Meetings in 1929–30

SECTIONS. *Ann Arbor:* May 22. *Boston:* November 5, February 3, May 27. *Cleveland:* May 14. *Chicago:* December 16, February 26, May 8. *Halifax:* October 21, May 2. *New Haven:* March 29. *New York:* December 5, March 13. *Philadelphia:* October 30, November 13. *Pittsburgh:* November 4, December 2, January 6, February 3, March 3, April 7, May 5, June 2. *San Francisco:* October 4. *St. Louis:* September 12, October 10, November 14, December 12, January 9, February 13, March 13, April 10, May 8, June 12. *Toronto:* October 2, January 7, February 20. *Vienna:* September 21. *Winnipeg:* April 23.

ASSOCIATION: *Eighth general meeting*, Toronto, March 24–26, 1930.

Meetings in 1930–31

SECTIONS. *Chicago:* October 16, January 8, February 1. *Halifax:* November 28. *Minnesota:* October 27, November 24, January 26, March 30, April 27. *New York:* December 10. *Philadelphia:* October 13. *Pittsburgh:* November 3, December 1, January 5, February 2, March 2, April 6, May 4, June 1.

ASSOCIATION: Ninth general meeting, Memphis, March 21–22, 1931.

All persons interested in dental research are invited to attend the scientific meetings of the Sections and of the Association.

INFORMATION FOR PROSPECTIVE SUBSCRIBERS OR CONTRIBUTORS

THE JOURNAL OF DENTAL RESEARCH is dedicated to the advancement of research, and to the dissemination of knowledge, pertaining to the teeth and mouth and to their relations to the body as a whole. It publishes original investigations and reviews in these fields, and in the sciences allied with or applied to them. Issued bi-monthly, at the *ends* of the months of February, April, June, August, October and December, each volume contains at least 600 pages, for which the subscription price is $5.00.

This JOURNAL is the official publication medium of the dental organizations named on the title page of this cover. Although without a "policy," the JOURNAL represents an unwavering purpose to keep widely open the channel to freedom of *responsible* and *respectable* expression of truth and opinion, by individuals or organizations, without qualification by or for any views its editors individually may entertain. In accordance with this purpose, papers contributed by societies are controlled and edited by the respective associations through their official representatives.

THE JOURNAL OF DENTAL RESEARCH circulates widely among the most enlightened and progressive dentists and physicians, and among the leading libraries, thus assuring the degree and quality of publicity for its contents that the best dental literature merits. Therefore there can be no important occasion, except in very special instances, for the detailed presentation elsewhere of a paper published in this JOURNAL. Accordingly, to prevent waste of the JOURNAL'S resources in needless duplicate publication, and also to lighten the labors of students of the literature of dentistry, papers in this JOURNAL are not published in, or reprinted from, other dental journals, except for unusual professional reasons.

Authors of papers *on original research* will receive, free of charge, 50 reprints of their papers, *if they indicate their desires before the issue containing them goes to press.* Copies in any number may be obtained at prices that will be quoted on request.

Address all communications relating to editorial matters to the JOURNAL OF DENTAL RESEARCH, 632 West 168th Street, New York City; business communications, to the JOURNAL OF DENTAL RESEARCH, Mount Royal and Guilford Avenues, Baltimore, Md.

CONTENTS

December, 1930

Preliminary comment on the Oral Hygiene Committee's criticism of the American Child Health Association's Research Monograph Number III, entitled "Public health aspects of dental decay in children." *George T. Palmer*, Dr.P.H. 655

A review of dental literature on pits and fissures. *Thaddeus P. Hyatt*, D.D.S., F.A.C.D. 659

Medico-dental case record: Ninth Report. *Bissell B. Palmer*, D.D.S., F.A.C.D., and *Malcolm W. Carr*, D.D.S 675

Oral infections and their relations to diseases of the eye. A symposium. *William F. C. Steinbugler*, M.D., *Henry W. Gillett*, D.M.D., F.A.C.D., and *Henry S. Dunning*, D.D.S., M.D., B.S., F.A.C.S. 685

Physico-chemical studies on the structure of dental enamel. III. A method for determining the rate at which a salt may penetrate the enamel. *Henry Klein*, D.D.S., Sc.D. 727

The effects of strontium administration on the histological structure of the teeth of rats. *Henry Klein*, D.D.S., Sc.D., *J. Ernestine Becker*, B.S., M.A., and *E. V. McCollum*, Ph.D., Sc.D. 733

Cysts in the incisor canal. Further studies of pathologic conditions in the region of the naso-palatine (incisor) canal and papilla palatina. *Joseph Schroff*, M.D., D.D.S. 739

Subperiosteal abscesses arising from the mandibular first molar. *Hugh W. MacMillan*, D.D.S., M.D., F.A.C.D. 763

William J. Gies Fellowship in Biological Chemistry at Columbia University. *Edgar G. Miller, Jr.*, Ph.D., Chairman of the Fellowship Committee of the Department of Biological Chemistry 771

Supplement:

I. New York Academy of Dentistry. Notes from the minutes of the proceedings in 1929–30. *Malcolm W. Carr*, D.D.S., *Editor* 775

II. William Jarvie Society for Dental Research. Notes from the minutes of the proceedings in 1929–30. *Joseph Schroff*, M.D., D.D.S., *Editor* 781

III. International Association for Dental Research. Memoranda on the proceedings in 1929–30. *William J. Gies*, F.A.C.D., *Secretary* 782

IV. Important books recently received by the *Journal of Dental Research* 786

Index 791

General matter for Volume X:

Announcements iii

In memoriam iv

Contents of the Volume v

WAVERLY PRESS, INC.

BALTIMORE, U. S. A.